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# HEALTH OF WORKERS IN . . .

# Chromate producing industry

FEDERAL SECURITY AGENCY

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# HEALTH OF CHROMATE WORKERS

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HEALTH OF WORKERS IN . . .

# *Chromate producing industry*

## *a study*

FEDERAL SECURITY AGENCY
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# FOREWORD

Within the past half century, the Division of Occupational Health of the Public Health Service has engaged in studies of potential health hazards in major American industries. As the possibility of harmful exposures to industrial substances and processes has become evident, the Division has frequently been requested by management and labor to conduct studies of this type.

In recent years, suspicion has steadily increased of carcinogenic hazards in the chromate-producing industry. Although various theories have been advanced as to the causative agent believed to be responsible for bronchiogenic cancer in the industry, this agent has not been precisely identified.

In the absence of a comprehensive clinical and environmental study, the Public Health Service was requested by the industry to undertake such an investigation. The results of this study constitute a contribution to knowledge in that they substantiate the existence of a cancer problem among chromate workers, help to identify the causative agents, and present methods of controlling the hazard.

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# ABSTRACT—SUMMARY

This report presents the clinical and environmental findings of an investigation to determine the present health status of chromate workers and to evaluate the effects of the working environment on their health.

Environmental investigations were made in six plants which are directly involved in the manufacture of chromates and bichromates from chromite ore. These plants are located in the states of Maryland, New Jersey, Ohio and New York and employ a total of approximately 1,200 persons. The same basic raw material, chemical grade chromite ore ($FeO \cdot Cr_2O_3$), is being processed by all of the plants.

Basically, the manufacturing process in all of the plants is the same. Briefly, it consists of roasting the finely ground chromite ore with soda ash or with a soda ash and lime mixture to produce a water soluble chromium compound, sodium chromate, which is converted by acidification and crystallization into crystalline sodium bichromate. Soluble materials other than chromates and bichromates occur in the chromate and bichromate liquors. By-products such as alumina, sodium sulfate, and sodium vanadate may be recovered from these liquors.

Approximately 1,600 samples of airborne materials were collected with the standard impinger and the midget impinger. About 100 material and settled dust samples were also analyzed.

It was found that the dry-end processes generate dusts containing chromite ore, soda ash, roast, residue and sodium chromate. Sodium bichromate and sodium sulfate are usually found associated only with the wet-end processes.

An appreciable portion of the total chromium was found to be present in an acid soluble-water insoluble state, indicating the presence of a form or forms of chromium which are dissimilar from either insoluble chromite ore or water soluble, hexavalent chromium. These acid soluble-water insoluble chromium compounds were found to be associated especially with roast or residue processes, although they were noted in many other parts of the chromate plants.

The greatest weighted average exposure to chromium-bearing dusts occurred in the dry end with maximum mean exposure of 1.07 milligrams of total chromium per cubic meter of air for mill room laborers. The greatest weighted average exposure (0.17 mg $Cr/m^3$) to hexavalent chromium occurred among potash production operators. Occupations with average exposures greater than 0.10 mg of water soluble hexavalent chromium per cubic meter of air included residue mix operators, crane operators, leach operators, sulfate recovery operators and centrifuge operators. The two occupational groups which process residue had the greatest average exposure, excluding tanning compounds, to acid soluble-water insoluble chromium. These groups are residue mix operators (0.17 mg $Cr/m^3$) and residue mill operators (0.15 mg $Cr/m^3$).

A medical examination was made of 897 chromate workers. Among this group were 10 persons who were considered as having bronchiogenic car-

cinoma. Of these, 8 had, in addition to roentgenological and clinical findings, histological evidence (pneumonectomy in 5 cases, biopsy in 1 case, necropsy in 1 case, and bronchial washings Class V in 1 case). The other 2 had, in addition to roentgenologic evidence of bronchiogenic carcinoma at the time of the survey, extension of the lesion in subsequent films and clinical findings of bronchiogenic carcinoma. The mean age of the 10 workers was 54.5 years and the mean exposure to chromates was 22.8 years. This experience gives a rate for bronchiogenic cancer of 1,115 per 100,000 persons for chromate workers which is far above that found among a comparison group.

A study of the morbidity and mortality experience of male members of sick benefit associations in seven chromate-producing plants showed a great excess of cancer of the respiratory system. There were nearly 29 times as many deaths from respiratory cancer among chromate workers as would be expected based on the experience of all males in the United States. The respiratory cancer mortality rate for colored males was more unfavorable than for white males. The total rate for cancer at all other sites was not higher among chromate workers.

Perforation of the nasal septum was found among 56.7 percent of the chromate workers. It was noticed that the colored chromate workers had a greater prevalence of nasal perforations than the white chromate workers. They also developed their perforation in a shorter period of time.

There is no evidence to show that exposure to chrome compounds affected the rate of dental caries attack. Some chromate workers developed a yellowish discoloration of the teeth and tongue. A higher percentage of chromate workers experienced gingivitis and periodontitis.

Pulmonary markings suggestive of fibrosis were not important among chromate workers, but bilateral hilar enlargements were noted.

Correlation of the clinical and environmental findings suggests possible factors in the working environment which might influence the abnormally high rate for bronchiogenic cancer among chromate workers. Previous investigators who considered the hexavalent chromium compounds to be harmful have stressed their chemical and physiological activity, minimizing their extreme solubility and consequent rapid dissipation. On the other hand, advocates of the trivalent chromium compounds have stressed their prolonged retention, minimizing their possible lack of physiological activity, due to their extreme insolubility. This report introduces for consideration other derivatives of chromite ore, namely, the acid soluble-water insoluble compounds. These acid soluble compounds are produced in the roast and remain principally in the residue. Consequently they are present in appreciable amounts wherever roast or residue is encountered.

Based upon the findings of this study the following recommendations are presented: The problems involved in the control of the dusts in this industry are not unusual but are generally typical of the chemical industry and require the application of established industrial hygiene engineering principles. Facilities and competent personnel should be provided to perform routine air analyses throughout each of the plants. Adequate dust control features should be incorporated in the design of all new equipment and in the redesign of old equipment. More complete enclosure of process equipment and conveying systems as well as greater use of local exhaust ventilation should be practiced. Housekeeping should be perfected to prevent accumulation of dusts and spillage. Until air concentration can be reduced to a safe level in certain areas or special operations, personal protective devices should be used.

It is recommended that all employees who have worked 5 years or more in the chromate industry should be X-rayed every 3 months and their films should be read by a competent roentgenologist. The study of the morbidity and mortality experience of workers in chromate plants should be continued. The local health department should follow up all chromate workers who have worked in the industry 5 years or more.

# ORIGIN OF THE STUDY

The German medical literature, prior to World War II, contains several articles relating to chromates as a possible cause of the bronchiogenic carcinoma cases observed in producing plants in that country *(66, 67, 68, 69, 70)*. The conclusions that chromates should be considered as carcinogenic were not generally accepted by the medical profession in the United States. However, in 1945 a death from bronchiogenic carcinoma, alleged to have been caused by exposure to chromates in a chrome-producing plant in this country, emphasized in the minds of the executives of that company the importance of a thorough investigation.

The need for such a study was stressed by W. C. Hueper, now of the National Cancer Institute, Public Health Service, who recommended that the company review the experience of chromate plants in Europe.

During the year 1946, an officer of the company visited the German chromate plants in occupied areas and made a personal investigation of the medical records of the German chromate producers. He had the opportunity to consult with the plant officials and physicians in Germany, who were of the opinion that chromates are carcinogenic.

The industry then consulted Leroy U. Gardner, director of the Saranac Laboratory for the Study of Tuberculosis, Saranac Lake, New York, who had been recommended as the outstanding authority in this country in matters relating to the effect of inhalation of dust and pulmonary disease resulting from industrial exposure.

Upon Gardner's death in October, 1946, the industry consulted A. J. Lanza, then assistant medical director of the Metropolitan Life Insurance Company, a former officer of the Public Health Service, and an authority on industrial medicine.

About the same time, Anna M. Baetjer, associate professor of physiological hygiene, School of Hygiene and Public Health, The Johns Hopkins University, Baltimore, Maryland, was engaged by one of the chromate producing companies to review the medical literature with respect to the relationship between exposure to chromates and the incidence of pulmonary carcinoma. In May, 1948, Baetjer submitted such a review to the industry which was published in November, 1950 *(110)*.

Upon the recommendation of Lanza, executives of all chrome-producing plants in the United States were requested to attend a meeting, at which time they were advised of the German experience and the possibility of a relation-

ship between exposure to chromates in chrome-producing plants and the incidence of pulmonary carcinoma. This meeting occurred in 1947.

Willard Machle of New York was employed by the industry to gather data on cases of bronchiogenic carcinoma that had occurred in the industry over the preceding ten years. W. M. Gafafer of the Public Health Service was consulted by Machle, and cooperated in the analysis and presentation of the data. The report was submitted to the industry and published on August 27, 1948 *(35)*.

All member companies of the industry called upon the Surgeon General of the Public Health Service in 1948 advising him of their suspicions and of the investigation initiated by the industry. At this time a request was made of the Public Health Service to make a survey of the industry with the following purposes: (1) To determine the relationship of exposure of employees to the incidence of bronchiogenic carcinoma; (2) to recommend a program of medical control; and (3) to recommend a program of engineering control.

Member companies promptly advised state departments of health and labor of the result of their investigations and solicited state assistance in the solution of their problems. To that end, industrial physicians, hygienists and engineers were consulted in regard to control methods in all producing plants.

For the past several years, the industry has altered manufacturing processes to the end that proper engineering practices and hygiene control measures may be established for the control of hazards, and that medical supervision and care may be effective for the early detection and treatment of bronchiogenic carcinoma.

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# REVIEW OF THE LITERATURE

Shortly after the chromate-manufacturing industry came into existence, reports began to appear in the medical literature concerning the effects of chromates on the health of the workers.

In 1827, Cumin *(1)* of Scotland described the presence of large penetrating ulcers on the hands of two men who worked in a local dye works and in the course of their work frequently dipped their hands and forearms into a solution of potassium bichromate.

In 1833, in Baltimore, Ducatel *(2)* cited the occurrence of deep skin ulcers of the hands and arms among workers in a chromate-producing plant.

In 1869, Delpech and Hillairet *(3)* investigated a factory in France which made potassium chromate and bichromate. They described and discussed dermatological lesions as well as the production of nasal septal ulcers and perforations.

As the production and use of chromium compounds became more widespread the medical literature on their effects on health grew considerably. The most numerous and widespread reports are those which describe ulcers rather than systemic toxicological effects due to chromium products.

In 1902, Legge *(4)* reported that of 176 workers in a Scottish bichromate-producing plant, 22.1 percent had unhealed chrome sores. A report *(5)* by the Medical Inspector of Factories of England for 1930 noted that of 223 persons examined in a chrome-plating works 42.6 percent had dermatitis, ulcers or scars thereof. Reports *(6)* in England revealed a steady increase of chrome ulceration of the skin in chromate workers: in 1933, 73 cases were reported; in 1938, 115 cases; and in 1939, 159 cases. In German bichromate plants employing about 700 men an incidence of 20.7 percent of occupational dermatitis was reported *(7)*.

In India, Naidu and Rao *(8)* reported an incidence of 23 percent ulcers and 33 percent scars in a bichromate-producing plant employing 152 workers.

In the United States, for the years 1928–1937, chrome ulceration of the skin caused by chromium compounds accounted for 3 percent of compensable occupational diseases reported in Ohio and a similar percentage was reported for Massachusetts for 1925–35 *(9)*. The skin lesions have been described by various authors *(1, 2, 10, 11)* as being round, nonspreading, deeply-penetrating ulcers which have a hard, well-defined, circular, heaped-up, usually noninflammatory periphery, with a central clean-cut cavity leading down to a base covered with exudate or a tenacious crust. The lesion begins as painless papules of pinhead size that gradually enlarge

to form the mature lesion which may vary from 3–10 mm in diameter *(12)*. The early literature *(1, 2)* describes cases in which the ulcers extended to remarkable depths, having burrowed through the arm or hand, and in some instances having involved the bones and joints *(13)*. These lesions, which are often labeled "chrome sores," "chrome holes" or "chrome ulcers," occur particularly where there is exposure to the hexavalent chromium compounds, namely, chromic acid or its salts, particularly the bichromate salts *(12, 14)*. The phenomenon is presumably due to the escharotic effects of free chromic acid which can be present as such or can be formed by the hydrolysis of chromium trioxide or the alkaline chromates. Thus, the relative importance of this problem in this industry, at least in the recent past, is apparent and is well documented.

Another local lesion, perforation of the nasal septum, like the cutaneous ulcer due to chromium compounds, is characteristic for this industrial exposure. Cumin *(1)*, in 1827, noted the presence of this lesion. In 1869, Delpech and Hillairet *(3)* described this pathological process among men exposed to monochromates and dichromates of potassium. They added further that they did not believe that chromite ore played any part in the process. In Germany many reports *(15, 16, 17, 18)* described the lesion and gave a prevalence varying from 35 to over 50 percent among chromate workers. This led to stringent regulations to govern the industry in Germany.

In 1902, Legge *(4)* found perforated nasal septa in 71.6 percent of 176 workmen and ulceration without perforation in 11.3 percent. A code of special rules was then set up to govern the industry in England. In 1916, Mitchell *(19)* reported finding a high incidence of perforation of the septum among workers in English chromate-producing plants.

In Italy *(20, 21, 22)*, in Russia *(23, 24, 25)*, in Spain *(26)*, in Sweden *(27, 28)* as well as in India *(8)*, similar reports were made.

In the United States MacKenzie *(29)* reported in 1884 on the toxic effects of chromates on the nose, throat and ear. He related that the workmen employed in the chambers where bichromate is made invariably acquire perforation of the cartilaginous portion of the septum, generally within a few days. In 1928 and 1929, several reports *(30, 31, 32, 33)* referred to nasal septal ulceration and perforation in workers engaged in chromium plating. Subsequent reviews of the chromate-producing industry *(34, 35)* report a similar incidence of this lesion.

The mucous membranes of the oral and upper respiratory tract have been reported as being attacked by the hexavalent compounds of chromium, and reports of ulceration and congestion of these structures have appeared. Lesions of the pharynx *(12, 32, 36, 37)*, soft palate *(12, 32, 38)*, tonsils *(36, 37)* and mouth *(12)* have been described. Bloomfield and Blum *(31)* have reported that a concentration of chromic acid greater than 0.1 mg per cubic meter of air is sufficient to injure the nasal mucous membrane. Mancioli *(39)* called attention to chronic pharyngitis associated with ulcera-

tion of the larynx and of the vocal cords in Italian bichromate workers. Wilensky *(23)*, in Russia found an incidence of pharyngitis of 41.7 percent in 278 bichromate workers whom he examined. Lieberman *(38)* described the throat of the chromate workers as being dry, red and having a glazed appearance.

Mancuso *(34)* noted that 86.6 percent of 97 workers in an Ohio chromate-producing plant had chronic chemical rhinitis, 42.3 percent had chronic chemical pharyngitis; that laryngitis, with hoarseness, occurred in 10.3 percent and congestion of the vocal cords, without hoarseness, in another 10.3 percent. Thickened sinus membranes were present in 27.0 percent of the workers and there was a total incidence of 11.8 percent of polyps in these structures, the majority occurring in those workers exposed to the soluble chromates.

Conjunctivitis and keratitis are frequently mentioned as occurring in this industry *(13, 37)*. Naidu and Rao *(8)* found in their study of 152 men in a chromate-producing plant that 10 percent of the workers had eye complaints referable to their chromate exposure.

Ducatel *(2)* as early as 1833 cites Gmelin as studying the acute toxic effects of chromates in animals. Lehmann *(15)* reported acute pulmonary deaths in cats as the result of inhalation of a chromate salt. Barbera *(40)* exposed guinea pigs to chromic acid fume and noted that the chromate was absorbed rapidly from the lungs, and that the latter were inflamed and hyperemic. Akatsuka and Fairhall *(41)*, in testing the toxicity of trivalent chromium salts, had cats breathe a high concentration of chromium carbonate. They found no evidence of ill effects in the animals. Meyers *(42)* in 1950 described two cases of acute chemical pneumonitis which occurred following exposure to high concentrations of chromic acid mist for periods of from one to four days. These men were acutely ill for two weeks with dyspnea, cyanosis and fever, and even after six months they still complained of cough and sharp, burning chest pain. One of the men had a pleural effusion which lasted for six months after the attack. Mancioli *(39)* found cough to be a common complaint among the 107 workers he examined in a sodium bichromate plant. Several observers *(25, 34, 43, 44)* have reported diffuse enlargement of the alveolar septa and a peculiar type of interstitial and interalveolar fibrotic process.

Reports of the occurrence of bronchial asthma following sensitization to compounds of chromium have been made on several occasions *(4, 45, 46, 47, 48, 49)*. Most of these cases have followed exposure to hexavalent chromate, although Broch *(47)* cites two workers who showed hypersensitivity to metallic chromium dust.

In general, the toxic effects from trivalent chromium compounds have been minimal because of their poor absorption. Chromic carbonate and chromic phosphate *(41)*, and chromic lactate *(50)* have been found to have little toxic activity in experimental animals. In fact, Schürch and his asso-

ciates *(51)* have demonstrated the efficacy of using trivalent chromic oxide as an index for determining digestibility of food.

The hexavalent compounds of chromium, on the other hand, are quite active. Many experimental studies *(2, 40, 41, 50, 51, 52)* as well as clinical studies *(53, 54)* have demonstrated this fact. Workers exposed to hexavalent chromium compounds as in chromium plating *(11, 38)* and in chromate production *(34)* have experienced gastrointestinal symptoms of anorexia, nausea and vomiting. The death of twelve persons from the use of an anti-scabetic ointment which had potassium bichromate as one of its components was a tragic illustration of hexavalent chromium toxicity *(55)*. In addition to the local cutaneous necrosis and symptoms of acute toxicity, nausea, vomiting, shock and coma, albuminuria and hematuria were constant findings. Acute nephritis developed with levels of nonprotein nitrogen as high as 268 mg percent. Renal hyperemia and tubular necrosis, with little glomerular changes were necropsy findings. Other reports *(46, 53, 56)* have demonstrated renal changes as the result of acute exposure to chromic acid and bichromate. Chronic exposure in chromium plating *(38)* and chromate production *(34)* has not demonstrated any abnormalties in the urine of these workers. Ophüls *(57, 58)* in his experimental studies, concluded that sublethal doses of bichromate must be given frequently and that it is difficult to produce lasting renal lesions with sublethal doses. Other experimental work *(59, 60, 61, 62)* has confirmed this finding.

Acute exposure to chromates has demonstrated an initial stimulation of red and white cells and platelet production, followed by a diminution of these elements *(55, 63, 64, 65)*.

Conflicting reports on the effects of chromates on the central nervous and cardiovascular systems have appeared. Brieger *(55)* noticed no definite central nervous system effects in the Breslau poisonings, whereas Thompson *(13)* reported in cases of chronic poisoning in humans the occurrence of headaches, tremors of the hands and tongue, spasm of the eyelids, and some difficulty of speech. With respect to the cardiovascular system, Ophüls *(57)* in his animal work found no damage from chromates to the heart and blood vessels, although hemorrhages into the endothelial linings (pleura, pericardium, peritoneum) were described *(55)* in the Breslau human cases. It is possible that these latter effects were due to the direct caustic action of the chromates from the overwhelming acute exposure.

The first report which described the occurrence of lung cancer in chromate workers was that by Lehmann *(66)* of Germany in 1932. In this report he described two cases of lung cancer as observed by Pfeil in 1912 in a chemical plant at Ludwigshafen. Lehmann found that chronic bronchitis was prevalent among workers in chromate production, but as to the question of pulmonary carcinoma he concluded that the cases so far reported were too few to be significant, that isolated cases occur in all classes of workers, and that the increase of cancer of all kinds is due partly to better diagnostic

methods, partly to the increased proportion of the elderly in the population and the successful lowering of other causes of death, especially tuberculosis.

In 1932, Teleky *(67)* at the request of the German Factory Workers' Union, investigated six suspected cases in a large chromate factory of which only one was cancer of the lung.

In 1935, Pfeil *(68)* reported five additional cases of lung cancer in the Ludwigshafen plant which had been closed since 1923. He did not state in which part of the plant these men had been employed, nor their occupations since 1923.

In 1938, Alwens and Jonas *(69)* reported a total of twenty cases of lung cancer among former workers in a chromate plant in Griesheim which was closed in 1931. Only ten of these workers were employed exclusively in the chromate-producing section of the plant; one worked in the aniline industry for five years preceding the diagnosis of cancer of the lung. The remaining ten worked only intermittently in chromate production and were exposed to other chemicals in other portions of the plant. The investigators believed these people had exposure to chromates because two had a perforated nasal septum. One of the cases later proved to be a primary carcinoma of the pancreas at necropsy. Later Alwens *(70)* reported one case at Nerdingen, one at Bitterfeld and possibly one at Leverkusen.

In 1937, Baader *(71)* reported a case of lung cancer in a patient who had applied pigment (compound not mentioned) by hand and by spray for many years.

In 1938, Gross *(72)* reviewed 39 cases and estimated that the German plants producing chromates had employed about 2,000 men since 1880, of whom 1,000 had worked in the chromate industry only a short period of time.

In 1943, Gross and Koelsch *(73)* reported eight cases of pulmonary carcinoma occurring in the chromate-color industry. Only one of these cases had necropsy findings, and two had no clinical data but were based on death certificates.

In 1944, Letterer, Neidhardt and Klett *(44)* reported two cases (with lung sections) of bronchiogenic carcinoma in workers exposed to lead and zinc chromates.

In 1947, Goldblatt and Wagstaff *(74)* reported ten workers with lung cancer at Leverkusen; seven had died and three were ill at the time of the survey.

Whereas, the German literature was first to suggest that some factor in the occupation of chromate workers was responsible for the development of bronchiogenic carcinoma, data to calculate rates are lacking. Many of the cases were not fully diagnosed, and a number of the cases were exposed to other substances, some of which may have been carcinogenic, such as anthracene and its derivatives, as well as the quinone compounds. The cases were reported sporadically and no figures were given of prevalence in the

chromate industry as compared with other industrial workers of the same age and sex.

These German reports of bronchiogenic carcinoma in chromate workers were the only ones to be found in the literature until recently, although articles on the occurrence of nasal perforation and dermatitis are abundant. Bidstrup *(75)* in 1951 reported finding cancer of the lung in a 59-year-old man among 724 workers she examined in the chromate-producing industry in Great Britain. She felt that her figures were too small for definite conclusions but that "a follow-up study over several years will provide data for which the true evidence of carcinoma of the lung in workmen in the chromate-producing industry in Great Britain may be assessed."

Koelsch *(76)* reported knowing of one case among Swiss chromate workers. No further details were given.

The first study of lung cancer in chromate workers in the United States was by Machle and Gregorius *(35)* in 1948. They analyzed the mortality data from the records of the group life insurance policies of chromate companies. Death from cancer of the respiratory tract was reported to have occurred in 42 employees from seven plants in the United States between 1937 and 1947. The crude death rate for cancer of the lung was 25 times the normal. The criterion for diagnosis was records of necropsy, biopsy, positive X-ray, or "other valid clinical data." Baetjer *(77)* in a subsequent study of hospital records concluded that the percentage of lung cancer patients who had been exposed to chromates was significantly higher than would be expected on the basis of control groups.

Mancuso and Hueper *(78)* examined the records of employees who had worked one year or more between 1931 and 1949 in a chromate-producing plant in Ohio to determine the number who had died and the causes of death as listed on their death certificates. Of the 33 chromate workers who had died, six (18.2 percent) had a diagnosis of pulmonary carcinoma. For all males who had died between 1937 and 1947 in the county where the chromate plant is located, the percentage of all deaths due to cancer of the respiratory system was only 1.2 percent (34 deaths). A table showing years of exposure and other data on seven cases of lung cancer in chromate workers in this plant is given. One of these died outside the county and is not included in the above calculation.

Thus, other than the report by Machle and Gregorius *(35)* no review of the entire chromate industry has been undertaken in the United States. No clinical survey of the health status of the workers in the entire chromate-producing industry has been reported.

# THE CHROMATE-PRODUCING INDUSTRY

There are six plants in the United States which are directly involved in the manufacture of chromates and bichromates from chromite ore. At the time of the study one of these plants divided its processes between two units which were located several miles apart and, although two separate physical plants were involved, they have been considered as one plant for purposes of the engineering section of this report. The six plants are located in the states of Maryland, New Jersey, Ohio and New York, and employ about 1,200 persons, of which about 750 are production workers. The remainder are maintenance, supervisory and office workers.

The largest plant employs about 240 production workers and the smallest about 60. The total production is over 100,000 tons of sodium bichromate ($Na_2Cr_2O_7$) per year. One of the plants utilizes all of its own production for the manufacture of chromium pigments. Four of the others utilize a portion of their production for the manufacture of other chromium chemicals such as sodium chromate, potassium bichromate, ammonium bichromate, chromic acid and basic chromium sulfate.

The same basic raw material, chromite ore ($FeO \cdot Cr_2O_3$), is now being processed by all of the plants. It is obtained from the Transvaal, South Africa. In the past, ores had been obtained from New Caledonia, Turkey, Russia, Philippines, Cuba and domestic sources.

## *Description of Processes*

Basically the manufacturing process in all of the plants is the same. Briefly, it consists of roasting the finely ground ore with soda ash or with a soda ash and lime mixture to produce a water soluble chromium compound, sodium chromate ($Na_2CrO_4$), which is converted by acidification and crystallization into crystalline sodium bichromate. Soluble materials other than chromates and bichromates occur in the chromate and bichromate liquors. By-products such as alumina, sodium sulfate and sodium vanadate may be recovered from these liquors.

The process may be divided into two general phases known in the industry as the "dry end" and the "wet end." The dry end of the process may be subdivided into segments of operation referred to as milling, roasting and leaching. The wet end of the process may be subdivided into segments of operation referred to as neutralizing, treating and concentrating. Although basically these processes are the same, modifications are prevalent throughout the industry. Mix proportions are different; the extent to which residue

is recycled varies; methods of roasting and leaching differ; methods of concentration vary, and many differences in minor procedures exist.

Subsequent to the completion of the engineering field work for this study two of the plants have made very extensive alterations in their manufacturing processes. Although these processes are basically the same as described, marked changes have been made in procedures and equipment in a commendable attempt to control the contamination of the working atmospheres. The new procedures are not dealt with in the following process description; rather the procedures and equipment in use at the time of the study are described.

Figure 1 shows a simplified flow diagram of the general processes found in the industry.

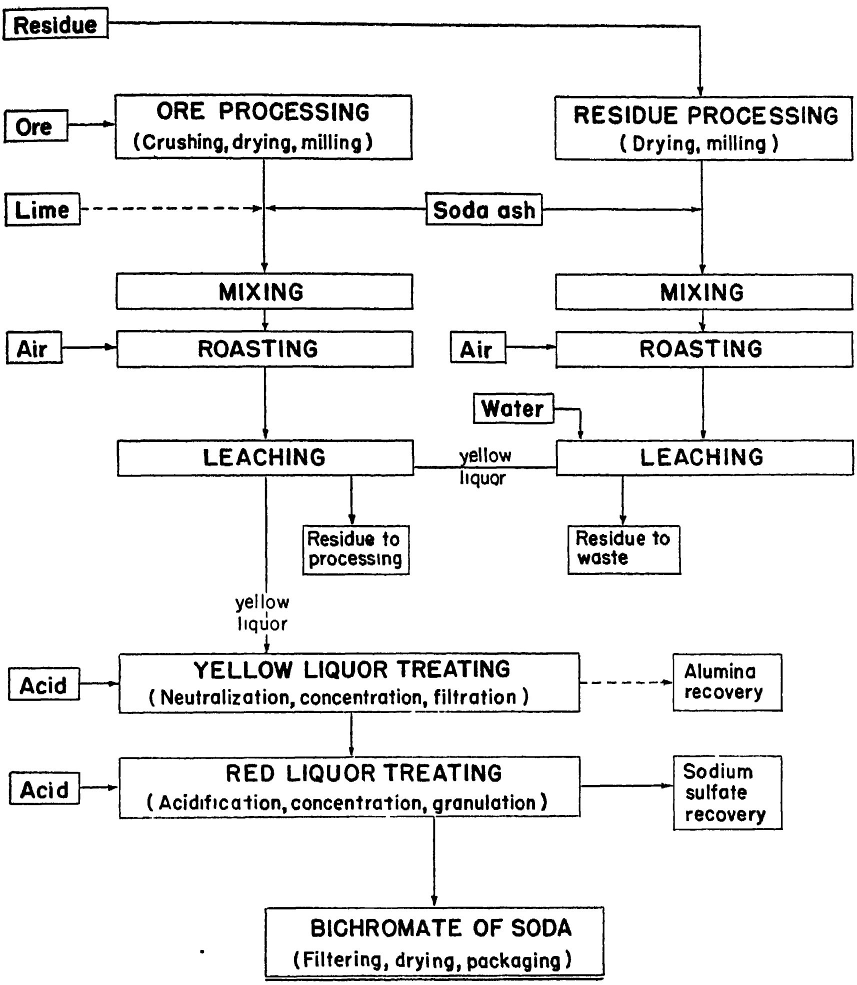


**FIGURE 1.**—Flow diagram of general processes used in chromate-producing industry in the production of sodium bichromate. (Dotted lines indicate presence in some processes only.)

### Milling

The ore is passed through a jaw crusher, dried in an oil- or coal-fired rotary, or rake, drier and pulverized in a ball mill to less than 100 mesh size. Weighed portions of the pulverized ore, soda ash or soda ash and lime, and finely ground filter residue from the plant's leaching operation are blended in a rotary mixer and conveyed mechanically to storage bins in the kiln area. The recycled residue present in the mix acts as a diluent and prevents sticking when the mix is roasted in rotary kilns.

### Roasting

The mix is slowly fed into direct oil-fired, rotary kilns. At the time of the study one of the plants pelletized the mix, and roasted these pellets in small (5 foot diameter) grates which were fired by individual oil-fired, reverberatory furnaces placed over the grates. The roasting is carried out in an oxidizing atmosphere at a temperature of from 1,800° to 2,200° F. depending upon the composition of the mix. The mix does not fuse but the molten soda ash reacts with the chromite to form the water soluble sodium chromate. If lime is present, it reacts with the aluminum components of the ore and subsequently prevents the solution of aluminum compounds when the roast is leached. The hot roast spills out of the lower end of the kilns onto an open mechanical conveyor system which transports it to storage or directly to the leaching equipment. Where pellets were used, the roast was passed through a roll crusher prior to the leaching process.

### Leaching

The roast is placed in one of a series of leach tanks which are large boxes with filter bottoms. Each filter box or leach tank usually holds about 125,000 pounds of roast, and when it is filled, leaching liquor is pumped through it. A counter-current leach process is employed, liquor being pumped from tank to tank so that a near saturated solution is obtained. Since the roast is hot and an exothermic reaction occurs when the solution is pumped onto it, steam is formed and often very vigorous boiling occurs for a considerable period of time after the hot roast has been completely covered with leaching solution.

The liquor resulting from the leach process is deep yellow and is known as "yellow liquor." It is a nearly saturated solution of sodium chromate and contains other water soluble materials such as sodium aluminate, sodium carbonate, sodium hydroxide and sodium vanadate, which may be present in the roast. The solid material remaining in the filter boxes following the primary leaching operation is known as "residue" or "mud." This residue contains a quantity of chromium compounds sufficient to make reprocessing economical. The residue is removed from the primary filters, dried in a continuous oil-fired or waste-heat-fired rotary drier, crushed and ground to 100-mesh size, mixed with soda ash, roasted, and leached in a process essentially the same as the primary (ore) cycle. A portion of the dried and ground residue is recycled for use in the primary mix.

Modifications of the leach process exist. One plant utilizes a continuous, rotary, counter-current extractor, discarding the fines carried with the liquor and recycling the coarser material. Another plant utilizes rake classifiers and continuous rotary filters. Another uses a wet mill in conjunction with continuous rotary filters.

### Neutralization

If sodium aluminate is present in the yellow liquor, the liquor is pumped into treat tanks where sulfuric acid is added and the hydrated alumina is precipitated at a pH of about 8. This neutralization may be finished with recycled, red mother liquor. The hydrated alumina is removed by continuous rotary filters or plate and frame presses. In some plants, the hydrated alumina is discarded, but in others it may be dried and sold with a yellow discoloration due to a small percentage of sodium chromate. The yellow liquor is sometimes marketed as a strong sodium chromate solution.

### Treat

The strong sodium chromate (40° Bé.) is treated with sulfuric acid (60° Bé.) to form sodium bichromate and sodium sulfate. Usually this acidification takes place in large (5,000 gallon) semi-enclosed boiling kettles.

The resulting solution is a deep red and is termed "red liquor" by the industry. The red liquor is concentrated and the sodium sulfate precipitates out of solution. This sodium sulfate is filtered out, washed and dried, and sold as an impure salt cake to the kraft paper industry. It may contain up to about 2 percent sodium bichromate. At one of the plants, however, the sulfate is redissolved and treated first with sulfur dioxide to reduce the remaining chromium and then with sodium carbonate to precipitate hydrated chromium oxide. The oxide is filtered off and the clear sulfate solution is then concentrated. The resultant sodium sulfate crystals are centrifuged, dried and sold as a pure sodium sulfate to the pharmaceutical and dye industries.

### Concentration

The red liquor is further concentrated in boiling pans or evaporators to about 66° Bé. and then pumped into large granulator tanks where the agitated solution is allowed to cool slowly. The tanks are water and air cooled. A granular product, $Na_2Cr_2O_7 \cdot 2H_2O$, forms as a thick slurry which is centrifuged and washed. The crystals are then dried in a rotary steam-heated drier and packaged in bags or drums. A 70-percent solution may also be produced from the liquor for marketing.

The concentrated red mother liquor may be recycled to the concentration tanks or used in the manufacture of potassium bichromate or tanning compounds. If it must be continually recycled, the concentration of undesirable impurities may build up to the extent that the product is affected adversely. Small amounts of chlorides apparently affect crystal formation and the

chloride concentration must be reduced in some mother liquors at intervals by electrolysis.

### Other products

Figure 2 shows the simplified flow diagrams of the processes used by some plants in the making of other products.

### Vanadium

Several of the plants recover vanadium from the sodium bichromate mother liquor by further acidification with sulfuric acid and filtering off the precipitated polyvanadates. This precipitate is washed, dried and drummed for marketing.

### Chromic acid

Two of the plants use a portion of their sodium bichromate production for the manufacture of chromic acid ($CrO_3$). Sodium bichromate is mixed with sulfuric acid (66° Bé.) in oil-fired cast iron pots. The mixture is agitated and heated to form a molten reaction mixture. The heavier chromium trioxide ($CrO_3$) formed collects in the bottom of the pot leaving the sodium acid sulfate floating on the top. As soon as the reaction has gone to completion, the bottom of the pot is tapped and the molten chromium trioxide is allowed to flow over water-cooled rolls which produces the chromic acid flakes. It is immediately packed in steel drums.

### Sodium chromate

Two plants are engaged in the small-scale production of sodium chromate. It may be crystallized from the alumina-free yellow liquor or may be produced by mixing proper proportions of dry sodium bichromate and sodium carbonate and heating trays of this mixture in a steam chamber. The resulting cake is air dried, milled and packaged.

### Potassium bichromate

Two plants manufacture potassium bichromate. It is manufactured by reacting sodium bichromate mother liquor with potassium chloride. Crystallization of the potassium bichromate is allowed to proceed in large tanks or the crystallization may be brought about in evaporators. The crystals are centrifuged, washed, dried, and packaged in bags or drums. The mother liquor is nearly saturated with sodium chloride which precipitates on concentration. The salt is washed and discarded and the solution is recycled in the process.

### Tanning compounds

Two plants are engaged in the manufacture of mixtures which are known as "one-bath" tanning compounds. The active ingredient of these tanning mixtures, basic chromium sulfate, $Cr(OH)SO_4$, is produced by reducing a sodium bichromate-sulfuric acid mixture with glucose in lead lined tanks. A very vigorous reaction occurs, which is allowed to go slowly to comple-

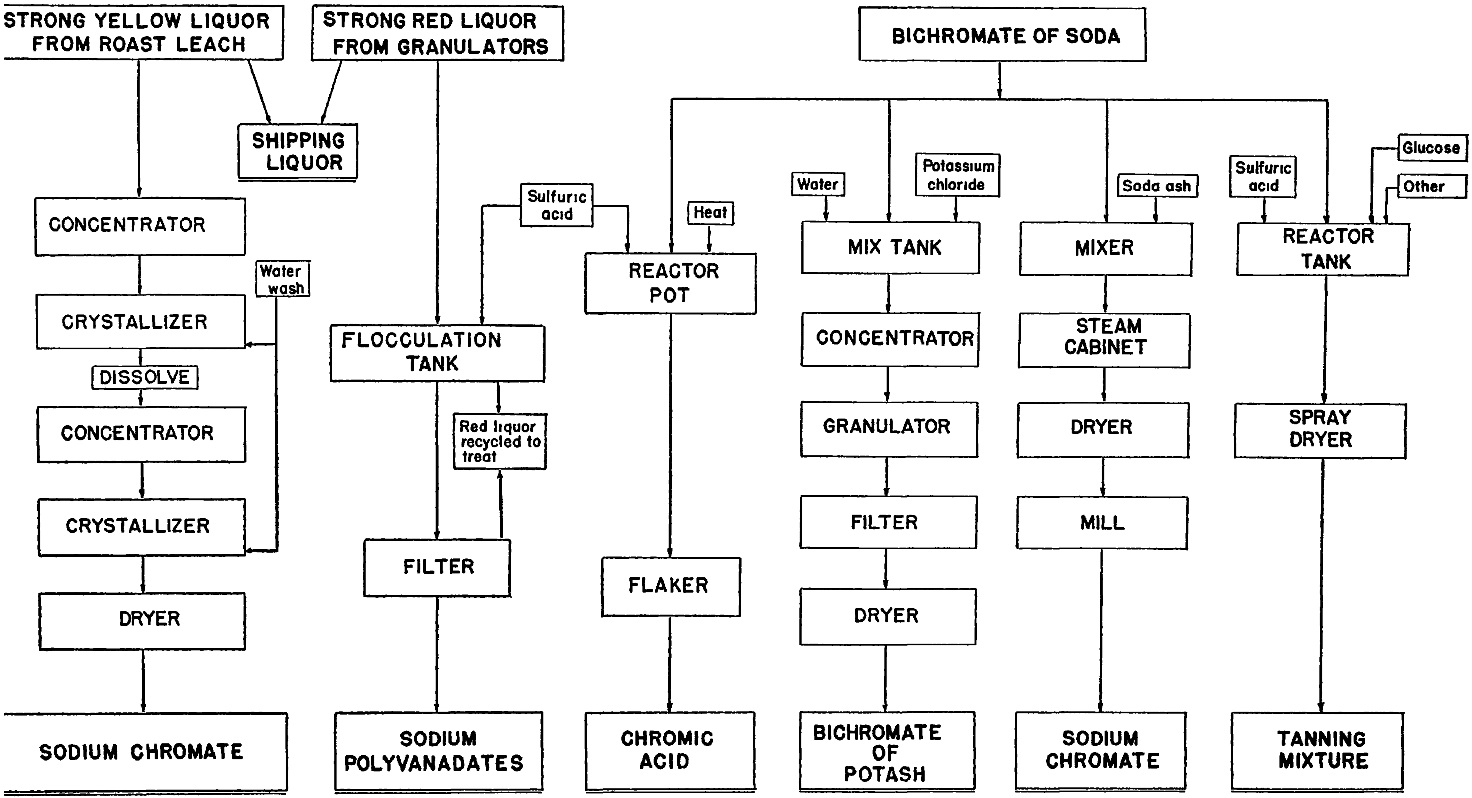

FIGURE 2.—Flow diagrams of processes used by some chromate producing plants in the making of sodium chromate and other products.

TABLE 1.—*Eight major functional groups and occupations associated with them.*

| Functional group | Occupational groups | General description of functions |
|---|---|---|
| Ore processing and Residue processing | Ore processors | Crush, dry, mill and convey ore in mill building. |
| | Lime and soda ash handlers | Convey lime and soda ash from railway cars to mill building. |
| | Mill laborers | Clean up spillage in mill building. |
| | Mix operators | Weigh ore, soda ash, residue and lime in mill building. |
| | Residue drier operators | Operate residue drier in kiln–leach area. |
| | Residue mill operators | Operate residue mill in mill building or kiln–leach area. |
| Roasting | Kiln operators | Operate roasting kilns in kiln–leach area |
| | Kiln building laborers | Convey roast and mix spillage, aid kiln operators in kiln–leach area. |
| Leaching | Crane operators | Operate overhead cranes, convey roast and residue in leach area. |
| | Leach operators | Operate leach filter pumps and related equipment in leach area. |
| | Mud operators | Maintain flow of wet mud in leach area to drier or dump. |
| Neutralizing and treating | Alumina recovery operators | Treat yellow liquor, filter alumina in alumina treat area, dry and/or bag alumina. |
| | Chemical treat operators | Treat yellow liquor with acid in liquor building. |
| | Sulfate recovery operators | Filter, dry, and convey sodium sulfate in liquor building. Also load railroad cars with sodium sulfate in area adjacent to liquor building. |
| Concentrating and granulating. | Liquor concentration and crystallization operators | Concentrate red liquor and operate granulators in liquor building. |
| Filtering, drying and packing. | Centrifuge operators | Centrifuge sodium bichromate slurry in liquor building. |
| | Bichromate drying and bagging operators | Dry sodium bichromate and pack bags or drums. |
| | Shippers. | Handle packaged materials in areas near soda packing. |
| Other products | Chromic acid cookers and packers | Feed sodium bichromate and sulfuric acid into cook pots, fire and drain pots, pack chromic acid flakes into drums. |
| | Potash production operators | Convert sodium bichromate to potassium bichromate. |
| | Tanning compound cookers, driers and packers | Mix and react acid–bichromate with glusoce, dry and bag tanning compounds |
| | Chromate operators | Produce sodium chromate. |

tion. Other constituents of the mixture are added and the resulting solution is dried in a spray drier. This compound, a green, finely divided material, is bagged and marketed under several trade names.

## Description of Occupations

Exact duplication of occupations is not generally found in the various plants. This is due primarily to variations in the processes, plant layouts, and quantity of production. A given workman's functions may consist of only a part of the group functions listed or may consist of several group functions. The former is more likely to occur in large plants and the latter in smaller plants.

Figure 3 shows the relationship of the eight major functional groups common to the industry according to a simplified flow diagram. Table 1 also shows these eight major groups in the general order of processing, but with their corresponding common occupations and functions.

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Three groups of workmen were selected for the purpose of comparison with the workmen in the chromate industry. The working environment for these men was studied for comparison with that of the chromate industry.

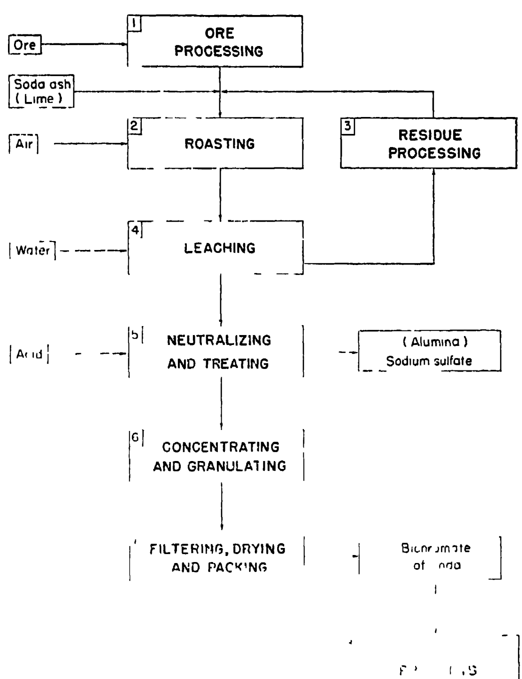


**FIGURE 3.—Flow diagram showing the eight major functional groupings of the chromate-producing industry.**

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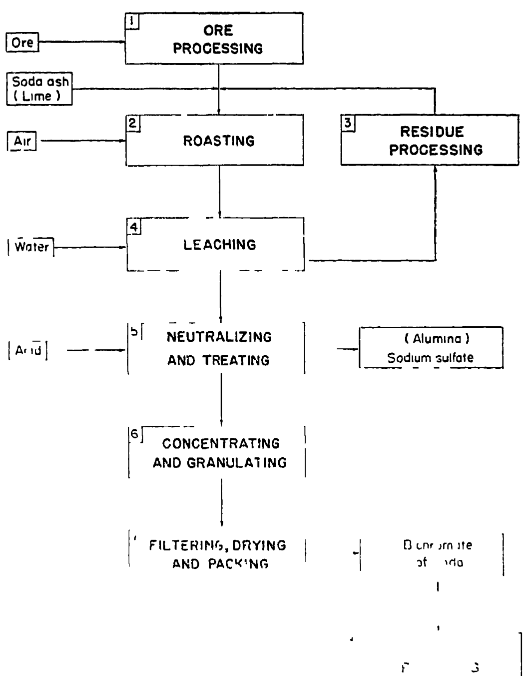


FIGURE 3.—Flow diagram showing the eight major functional groupings of the chromate-producing industry.

The plants in which these groups are employed are referred to as comparison plants A, B and C.

### Comparison plant A

This plant is engaged primarily in the manufacture of two products, namely, superphosphate fertilizer and silica gel. Only those workmen who were engaged in the manufacture of silica gel were included in this study group. All of this group were exposed to similar materials and the environment is therefore readily definable.

Silica gel manufacture consists of the treatment of silicic acid with sulfuric acid to produce a hydrogel which is subsequently washed, dried, sized, and then activated by heating. The finished silica gel is packaged for marketing.

### Comparison plant B

This plant produces paper products on which chrome pigments are used. It is located approximately a quarter of a mile from a plant which processes chromite ore. The settled dust at this plant is primarily paper fiber, but also contains a small amount of chromite ore which undoubtedly originated at the ore-processing plant.

### Comparison plant C

This plant manufactures refractory bricks. The process consists of crushing and drying refractory grade chromite ore, milling and grinding the ore, mixing and mulling the finely divided ore with magnesite wetted with sulfuric acid and then molding the mixture into bricks. Some of these bricks are then dried and shipped; others are slowly heated to a fusion temperature, cooled in a furnace, and then shipped.

# ENVIRONMENTAL STUDIES

## *Field Sampling Methods*

A total of about 1,800 samples were collected throughout the industry for the purpose of defining the atmospheric environment. Approximately 1,600 of these were samples of airborne materials and were collected with the standard impinger and the midget impinger. Distilled water was the collecting medium. About 100 material and settled dust samples were also collected in the various plants. The thermal precipitator, the high volume filter, the Zeiss konimeter, the NBS carbon monoxide indicator and the electrostatic precipitator were used occasionally in the study for the collection of special information.

Air samples were collected at each work location at random times on different days over a period of about two to three weeks in each plant. No effort was made to collect samples under extreme or unusual conditions. However, occasionally individual samples were collected when special or infrequent operations or special conditions warranted study. The number of samples collected at any one major working area was governed, to a large extent, by the variability of results obtained. Enough samples were taken to insure an accurate measure of average conditions. The initial phase of the study in each plant entailed air sampling at all operations in order to determine the variation of results and also to become familiar with the process. In the final phase the bulk of the remaining sampling time was devoted to those areas in which significant variations were found.

The duration of sampling ranged from about 10 minutes to several hours. About 1,100 air samples were of short duration; i.e., 10–30 minutes (0.1 cfm sampling rate) and about 500 air samples were of long duration; i.e., 30–160 minutes (1.0 cfm sampling rate).

Protective caps on the heads of the impingers and chemically clean extensions on the hose connections of the impingers were used to prevent the contamination of the sample. Clean pliofilm covers were kept on the impingers at all times before and after sampling. Exteriors of all impingers were washed down before the contents were submitted to analysis. These precautions were considered necessary because gross contamination of all exposed field equipment could not be avoided due, for example, to settling dust or splashing. Extreme precautions against contamination were also used in the field analytical procedures. The zero color standard, made up each day with the other color standards, served as a blank for the distilled water used as the collecting medium and also for the reagents used in the

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analysis. Also an occasional analysis was performed on the collecting medium from impingers which had been carried into the plants and returned without being used for air sampling.

The air samples were collected on all three shifts and analyzed for soluble hexavalent chromium the same day in the field. Portions of about one-third of all the atmospheric samples collected with the standard impinger were transferred to sample bottles and sent to the Cincinnati laboratories of the Division of Occupational Health for further analysis.

## *Analytical Methods*

The details of the analytical methods employed are described in Appendix.

## *Parent Materials*

The principal components of the airborne dust common to the industry are chromite ore, soda ash (and lime), roast dust, residue dust, sodium chromate, sodium bichromate, and sodium sulfate.

### Chromite ore

Chromite is a member of the mineral group spinel which is characterized by isometric crystallization and, therefore, also by isomeric optical properties. It is one of the first minerals to crystallize in a cooling magma, forming a crystalline, granular to compact mass, which varies greatly in composition depending upon the relative amounts of iron, chromium, magnesium, and aluminum present in the liquid mass. The theoretical formula of the mineral chromite is $FeO \cdot Cr_2O_3$, but the ores rarely conform. The iron may be replaced by magnesium and the chromium by aluminum and ferric iron. High grade ores, generally used by the metallurgical industry, contain more than 48 percent $Cr_2O_3$, while chemical grade ores such as those used by the chromate-producing industry may contain from 40 to about 45 percent $Cr_2O_3$. Refractory grade chromite ore may contain a lower percentage of chromium, but must be low in FeO and $SiO_2$. If the mineral contains less than about 10 percent chromium, it is considered to be a picotite mineral rather than a chromite.

Chromite ore, as observed in the chromate-producing and the refractory industries, is a hard, black, granular material before processing and is chemically very inert. A typical analysis of a chemical grade chromite ore is as follows:

| | *Percent* |
|---|---|
| $Cr_2O_3$ | 44.5 |
| $Fe_2O_3$ | 25.5 |
| $Al_2O_3$ | 14.2 |
| $SiO_2$ | 3.1 |
| MgO | 10.7 |
| $V_2O_5$ | 0.33 |

### Soda ash (and lime)

The soda ash used by the industry contains about 99.5 percent sodium

carbonate and the remainder is primarily sodium chloride and water. The lime which is used in some of the processes is pulverized, chemical grade, pebble lime.

## Roast

Fresh roast is a highly alkaline, partially fused, granular mixture of sodium chromate and other fusion products of the roast mix, unreacted ore and alkali. The composition as shown in table 2 varies and contains materials such as the oxides, silicates, chromates and chromites of iron, aluminum, magnesium, calcium, sodium and manganese, and some aluminates, and vanadates.

TABLE 2.—*Spectrographic analyses of dry-end materials collected in a plant using the no-lime process.*

| Material sample | Large amount [1] | Moderate amount [2] | Small amount [3] | Trace [4] |
|---|---|---|---|---|
| Ore | Cr, Fe, Mg | Si, Al | V, Mn | Na, Cu, Zn. |
| Mix | Cr, Fe, Na, Mg. | Al, Mn | Si, V | Ca, Pb, Cu. |
| Residue | Fe, Cr | Mg, Mn, Na, Al, Si. | | Zn, Pb, Ca, Cu, K, V. |
| Settled dust–mill building | Cr, Fe | Mg, Si, Mn | Na, Al | Zn, Pb, Ca, V, Cu. |
| Settled dust–roast area | Cr, Fe, Na | Mg, Al | K, Si, Mn, V | Pb, Ca, Cu. |
| Settled dust–kiln building | Cr, Fe, Na, Mg, Al. | Mn, Si | K, V | Zn, Ca, Pb, Cu. |

1 Large amount represents a quantity greater than approximately 10 percent.
2 Moderate amount approximates 1-10 percent.
3 Small amount approximates 0.05-1.0 percent.
4 Trace approximates less than 0.05 percent.
NOTE.—These samples were also examined for Cd, As, Ge, Tl, P, Ti, W, Bi, Sn, and Sb, and no traces were found.

Since the industry divides itself in one major respect, that is in the use of lime, two general types of roast (and residue) are produced. When chrome ore is roasted with soda ash alone, the alumina, silica, and vanadium combine with sodium to a large extent and under the influence of heat undergo recrystallization to form new combinations, some of which, such as the sodium aluminates and vanadates, are soluble in the leach liquor. Any alkali metal in the roast mix will probably effect some change of composition of the chromite ore when the mix is roasted. Calcium oxide, for example, which is present in some mixes, reacts typically as follows: $Cr_2O_3 \cdot FeO + CaO = Cr_2O_3 \cdot CaO + FeO$. The soda ash purposely mixed with the roast reacts in a similar manner and produces a chromite, which, in the presence of oxygen, is oxidized to the water soluble sodium chromate. Substantial amounts of the sodium also combine to form some of the products which are not soluble in the leach liquor. The altered chromites, although not soluble in the leach liquor, are far less inert than the chromite ore and are largely soluble in acid.

When chromite ore is roasted in the presence of lime, the alumina, silica, and vanadium react to a large extent, depending upon the proportion of lime used, to form water insoluble combinations. Calcium oxide may pro-

duce calcium chromate, as well as calcium chromite. The calcium chromate is unstable and at roast temperatures may give up part of its oxygen to form a calcium chromate-chromite complex. This complex, and also the altered chromites, are not soluble in water, but are soluble in acid. The distribution of chromium in various roasts and related materials is shown in table 3.

TABLE 3.—*Percent chromium, by solubility, of parent materials from various plants.*

| Parent material | Number of samples | Percent of total sample | | |
|---|---|---|---|---|
| | | Water soluble chromium | Acid soluble-water insoluble chromium | Acid insoluble chromium |
| Ore | 6 | 0.01- 0.07 | 0.01-0.04 | 29.6 -31.6 |
| Mix | 4 | 0.02- 0.52 | 0.02-0.13 | 4.4 -15.9 |
| Secondary mix | 1 | 0.88 | 2.66 | 2.5 |
| Roast | 6 | 1.11- 8.63 | 0-2.12 | 2.02-10.41 |
| Secondary roast | 2 | 4.14-12.3 | 0.79-1.1 | 0.16 |
| Residue | 3 | 0.4 - 0.75 | 0-2.7 | 2.4 - 9.15 |
| Secondary residue | 1 | 2.23 | 0.49 | 0 |

## Residue

The leach liquors remove a large part of the soluble alkalis, chromates, aluminates, and vanadates from the no-lime roasts. The leach is necessarily incomplete since some soluble material will remain imbedded within the granular pieces of roast. Subsequent reprocessing of the residue removes most of this soluble material before the final residue is discarded. The no-lime residues, then, are mainly mixtures of various combinations of $Fe_2O_3$, $MgO$, $SiO_2$ and lesser amounts of $Cr_2O_3$, and the remaining alumina and unreacted ore. A typical analysis of a dried residue from a no-lime roast leach is as follows:

| | *Percent* |
|---|---|
| $Cr_2O_3$ | 14.2 |
| $Fe_2O_3$ | 51.1 |
| $Al_2O_3$ | 9.2 |
| $SiO_2$ | 6.8 |
| $MgO$ | 11.3 |
| $CaO$ | 3.0 |
| Loss on ignition | 4.0 |

The results shown in table 4 indicate that most of the acid soluble-water insoluble portion of the total chromium present in the roast or residue is in the trivalent state. A close inspection of the percentages given in the table reveals that only about one percent of this portion of the chromium present in the roast sample and only about four percent of this portion of the chromium present in the residue was found to be hexavalent. This hexavalent chromium may be due to the chromite-chromate complex which is formed on the decomposition of some chromates.

A sample of the effluent leach slurry from a plant which did not use lime was filtered, and the residue was washed with water and analyzed for the acid soluble-water insoluble fraction and the acid insoluble fraction of the

chromium present. The ratio of the acid insoluble chromium to the acid soluble chromium was 3.5 mg to 4.42 mg. This indicates that about 56 percent of the remaining chromium in this particular no-lime residue was present in a state different from the ore.

TABLE 4.—*Percentage of total chromium and hexavalent chromium in samples of ore, roast and residue materials, according to solubility.*

| Solubility | Total chromium | Hexavalent chromium |
|---|---|---|
| | Ore | |
| Total | 31.34 | 0 |
| Water soluble | .02 | 0 |
| Acid soluble–water insoluble | .02 | 0 |
| Acid insoluble | 31.30 | 0 |
| | Roast | |
| Total | 14.10 | 9.13 |
| Water soluble | 9.10 | 9.10 |
| Acid soluble–water insoluble | 2.80 | .03 |
| Acid insoluble | 2.20 | 0 |
| | Residue | |
| Total | 5.50 | 0.50 |
| Water soluble | .40 | .40 |
| Acid soluble–water insoluble | 2.70 | .10 |
| Acid insoluble | 2.40 | 0 |

NOTE.—These samples were collected at a plant using the lime process.

The residue from the processes using lime retains most of the alumina and vanadium and only the soluble sodium chromate and soluble alkali are removed by the leach liquors.

## Sodium chromate

This is the desired product of the roast and also of the leaching liquor. Except where sodium chromate is purified or prepared from the bichromate for marketing, it does not exist in a pure state, but always occurs in mixed solutions, crystals, and dusts. In its pure state, however, sodium chromate forms deliquescent, yellow, rhombic crystals having the formula of $Na_2CrO_4$. This changes to the alpha form at 780° F. and melts at 1,460° F. It forms hydrates with 10, 6 and 4 molecules of water. Also, it is isomorphous with sodium sulfate.

## Sodium bichromate

Sodium bichromate (or dichromate) is the major product of the industry. The product is about 99.5 percent pure $Na_2Cr_2O_7$, the balance being principally sodium sulfate and a small amount of sodium chloride. It forms very deliquescent, orange-red monoclinic crystals.

## Sodium sulfate

Large amounts of sodium sulfate are continually separated from the bichromate liquors and subsequently processed. The sodium sulfate pro-

duced is impure and contains enough hexavalent chromium to give a yellow discoloration to the product. The amount of chromium remaining varies and depends upon the efficiency of the wash.

## *Airborne Materials and Settled Dusts — Composition*

The dry-end processes generate dusts containing chromite ore, soda ash (and lime), roast, residue, and sodium chromate. The dry-end operations are generally found to be adjacent to each other and in the same building or in adjoining buildings. Therefore, the airborne dust in the dry end is found to be a mixture of the dusts which have originated from the various processes present. Table 2, previously referred to, shows the results of the spectrographic analyses of several material and settled dust samples collected from dry-end sources in one plant. The relative amounts of the constituent elements are quite similar in all of the materials.

Sodium bichromate and sodium sulfate are usually found in connection with only the wet-end processes. Wet-end processes are associated with acid solutions while dry-end processes are associated with alkaline materials and solutions. In most instances, the physical plants are separate for the wet end and the dry end. Usually one or two buildings house the milling, roasting, and leaching processes and a separate building houses the treat, concentration and granulation processes. Hence, a given working area is principally affected by related wet or dry processes and less so by the more remote processes.

The major deviation from this general situation occurs in several plants where part or all of the wet-end function "neutralization and treating" is carried out in the same building as the dry-end processes. The general air pollution load undoubtedly consisted of chromates, bichromates, and associated components of the dusts throughout the plants. However, the contribution to the workroom air by the general outdoor air pollution was insignificant in proportion as demonstrated by comparatively very low air concentrations at outdoor and remote indoor areas.

Table 5 shows high percentages of acid soluble-water insoluble chromium for settled dust samples from many areas not associated with roast or residue processes. However, in settled dust samples, the water soluble hexavalent chromium originally present in the air has had considerable opportunity to react with and be reduced by other components of the settled dust. The reduction of the hexavalent chromium to a trivalent form undoubtedly occurs to some extent wherever it coexists with reducing dusts, and this reduced chromium contributes to the acid soluble-water insoluble fraction of the air samples when such dusts are once again airborne. Referring to table 5, the settled dust sample from the chromic acid area is an extreme example of this apparent occurrence. The air concentration of total chromium in this area is comparatively low. Also the amount of diluting,

duced is impure and contains enough hexavalent chromium to give a yellow discoloration to the product. The amount of chromium remaining varies and depends upon the efficiency of the wash.

## *Airborne Materials and Settled Dusts — Composition*

The dry-end processes generate dusts containing chromite ore, soda ash (and lime), roast, residue, and sodium chromate. The dry-end operations are generally found to be adjacent to each other and in the same building or in adjoining buildings. Therefore, the airborne dust in the dry end is found to be a mixture of the dusts which have originated from the various processes present. Table 2, previously referred to, shows the results of the spectrographic analyses of several material and settled dust samples collected from dry-end sources in one plant. The relative amounts of the constituent elements are quite similar in all of the materials.

Sodium bichromate and sodium sulfate are usually found in connection with only the wet-end processes. Wet-end processes are associated with acid solutions while dry-end processes are associated with alkaline materials and solutions. In most instances, the physical plants are separate for the wet end and the dry end. Usually one or two buildings house the milling, roasting, and leaching processes and a separate building houses the treat, concentration and granulation processes. Hence, a given working area is principally affected by related wet or dry processes and less so by the more remote processes.

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unreactive dusts, such as the inorganic residue and ore dusts, is low. Thus the accumulation of settled materials in the chromic acid area is similar in composition to normal atmospheric dusts, but also contains the chromium in a highly reactive state. The results show that only 4 percent of the settled dust sample remained in the hexavalent state, while 80 percent was present as acid soluble chromium. Sixteen percent of the chromium in this dust was in the form of chromite ore.

TABLE 5.—*Percentage distribution of chromium, by solubility, in settled dust samples from various working areas.*

| Working area | Percent total chromium in settled dust samples | Percent of total chromium | | |
|---|---|---|---|---|
| | | Water soluble chromium | Acid soluble-water insoluble chromium | Acid insoluble chromium |
| Mill building | 20.66 | 0.5 | 2 | 97.5 |
| Kiln discharge | 9.62 | 63 | 3 | 34 |
| Kiln feed | 11.32 | 19 | 2 | 79 |
| Alumina recovery | 2.87 | 57 | 40 | 3 |
| Alumina treat | 13.48 | 71 | 17 | 12 |
| Potash production | 18.47 | 52 | 18 | 30 |
| Soda packing | 12.56 | 11 | 82 | 7 |
| Mill building | 23.20 | 0.5 | 0 | 99.5 |
| Pelletizer | 15.24 | 2.5 | 0 | 97.5 |
| Pellet loading | 9.93 | 8.0 | 0.9 | 91.1 |
| Roast crushing | 17.26 | 22 | 0 | 78 |
| Sulfate treat | 15.26 | 70 | 25 | 5 |
| Sulfate recovery | 8.18 | 76 | 14 | 10 |
| Sulfate drier | .80 | 76 | 24 | 0 |
| Primary mix | 11.92 | 2.5 | 8.5 | 89 |
| Primary kiln discharge | 7.57 | 35 | 30 | 35 |
| Sulfate shipping | 1.0 | 76 | 9 | 15 |
| Liquor concentration | 8.76 | 55 | 39 | 6 |
| Ore milling | 4.52 | 0.1 | 2.9 | 97 |
| Primary mix | 4.94 | 2 | 0 | 98 |
| Secondary mix | 6.52 | 34 | 37 | 29 |
| Primary kiln discharge | 28.01 | 30 | 8 | 62 |
| Primary kiln feed | 6.41 | 7 | 4 | 89 |
| Secondary kiln feed | 22.41 | 4 | 6 | 90 |
| Filter | 7.39 | 45 | 33 | 22 |
| Filter residue | 6.04 | 60 | 29 | 11 |
| Residue drier | 6.04 | 28 | 38 | 34 |
| Sulfate wringer | 6.56 | 11 | 56 | 33 |
| Sulfate drier–ore mill | .16 | 0 | 0 | 100 |
| Treat | 7.58 | 53 | 27 | 20 |
| Granulator | 20.95 | 64 | 30 | 6 |
| Soda bagging | 16.77 | 62 | 25 | 13 |
| Chromic acid | 11.09 | 4 | 80 | 16 |
| Mixing | 16.30 | 44 | 2 | 54 |
| Leaching | 6.84 | 59 | 2 | 39 |

Some of the concentrations of acid soluble-water insoluble chromium found in some of the wet end areas, such as at treat, granulator, packing and chromic acid working areas, were due in large part to the basic chromium sulfate tanning compounds which were being handled or processed in the same or nearby areas.

Table 5 also shows that the chromium fraction of the settled dusts varies from 0.16 percent to 28.0 percent of the total dust for all the working areas listed. The dry-end dusts average 12 percent chromium and the wet-end dusts average 10 percent chromium of the total weight.

While it was not found possible to distinguish the chromate from the

bichromate in the air samples collected, the results of the concentrations of hexavalent chromium are reported as primarily either a chromate or a bichromate dust according to its source. Measurements of the pH on the collected sample solutions aided in this designation for some samples. Since dry-end (chromate bearing) dusts are very alkaline, many such samples were found to have a higher pH than many samples associated with the wet-end (bichromate) dusts. Some samples, however, had intermediate pH values and, consequently, it was not attempted to classify them on the basis of high or low pH.

As has been observed an appreciable portion of the total chromium is present in a water insoluble-acid soluble state, indicating the presence of a form or forms of chromium which are dissimilar from either chromite ore or water soluble hexavalent chromium.

Table 6 shows the results of the spectrographic analyses of various material and settled dust samples from the three comparison plants. This table

TABLE 6.—*Spectrographic analyses of settled dusts and finished products from comparison plants.*

| Sample | Large amount [1] | Moderate amount [2] | Small amount [3] | Trace [4] |
|---|---|---|---|---|
| *Comparison Plant A* | | | | |
| Settled dust - gel plant K1 | Si | Cu, Mg, Al | P, Co, Ca, Pb, Mn, Mo, Fe, V, Ti. | Cr. |
| Settled dust - gel plant K2-K10 | Si | Cu, Al | P, Co, Ca, Pb, Mn, Mo, Mg, Fe. | Cr, V. |
| Settled dust - super phosphate plant. | Ca | P, Si, Mn, Al (Fluorine present). | Cu, Mo, Mg, Fe. | Pb, Cr, V, Ti. |
| Finished product - gel plant K1. | Si | | Mg, Al, Ti | Co, Cu, Ca, Fe, Cr. |
| *Comparison Plant B* [5] | | | | |
| Settled dust - sample book department | Fe | Al, Si | Mg, Cr, Ca, Cu, Mn, Pb. | Zn, Mo, Ba, Ti, V, Ni, Sn, Na. |
| *Comparison Plant C* [6] | | | | |
| Refractory grade chromite ore fines. | Cr | | Fe, Mg, Al, Si | Mo, Co, Ti, V, Ca, Ni, Cu, Mn, Pb, Sn, Na. |
| Settled dust - milling and grinding room. | Cr, Fe | Mg | Ca, Al, Si | Mo, Co, Ti, V, Ni, Cu, Mn, Pb, Sn, Na. |

1 Large amount represents a quantity greater than approximately 10 percent.
2 Moderate amount approximates 1-10 percent.
3 Small amount approximates 0.05-1.0 percent.
4 Trace approximates less than 0.05 percent.
5 This sample was also examined for Hg, Bi, As, Cd, Be, Ag, Au, P, K, W and Co, and no traces were found.
6 These samples were also examined for Hg, Bi, As, Cd, Be, Ag, Au, P, K, W, Ba, and Zn, and no traces were found.

shows the major component elements encountered in these industries and also a rough indication of the quantity of each. Table 7 shows the chromium content of the settled dusts from comparison plant A was less than 0.05 percent, from comparison plant B it was less than 1 percent, while from

comparison plant C it was greater than 10 percent. It also shows that essentially all of the chromium in the settled dust from plant C was acid insoluble, chromite chromium.

TABLE 7.—*Percent of chromium content, by solubility, in samples of airborne and settled dusts from comparison plants.*

| Solubility | Type of dust | |
|---|---|---|
| | Airborne dust | Settled dust |
| | Plant A | |
| Total | 0.00 | 0.001[1] |
| Water soluble | .00 | .00 |
| Acid soluble | .00 | .00 |
| Acid insoluble | .00 | .00 |
| | Plant B | |
| Total | 0.000–0.008 | 0.45 |
| Water soluble | .000– .002 | .01 |
| Acid soluble | .000– .002 | .07 |
| Acid insoluble | .000– .004 | .37 |
| | Plant C | |
| Total | 0.640–4.16 | 8.50–18.4 |
| Water soluble | .000– .00 | .00– .02 |
| Acid soluble | .000– .03 | .00– .07 |
| Acid insoluble | .640–4.13 | 8.50–18.3 |

[1] Spectrographic analysis showed presence of chromium in trace amount.

## *Airborne Materials—Concentration*

Air concentrations of hexavalent chromium are reported as milligrams of chromic acid anhydride ($CrO_3$) per cubic meter of air. These values are based upon the determinations made in the field. These results have been converted in certain tables to milligrams of chromium (Cr) per cubic meter of air and reported as such for the purpose of comparison with the other forms of chromium. The laboratory determined water soluble chromium on selected air samples and also upon the settled dust and parent material samples. It may be noted from table 4 that all of the water soluble chromium present in the roast and residue material samples was in the hexavalent state.

Air concentrations of chromite ore are reported as milligrams of chromic oxide ($Cr_2O_3$) per cubic meter of air. These results have also been converted in certain tables to milligrams of chromium (Cr) per cubic meter of air for the purpose of comparison with the other forms of chromium. These results are based upon laboratory determinations of acid insoluble chromium in samples selected from each working area. It will be observed that essentially all (99.88 percent) of the chromium in the chromite ore is acid insoluble. Only 0.12 percent of the chromium was found to be either water or acid soluble. This finding was possibly due to contamination of the ore pile which was sampled.

This property of acid insolubility served as a means of separating the

chromium occurring as chromite ore from the other major forms of chromium encountered in the industry. The acid insoluble fraction is referred to as chromite ore. This fraction may also contain some slightly modified chromite ore, but its composition is still such that it is relatively inert and is not attacked by the acid digestion. Also, the property of extreme solubility of the hexavalent chromium occurring as sodium chromate or bichromate served as a means of separating that fraction of the total chromium. The water soluble fraction is referred to as water soluble, hexavalent chromium.

Results of air samples collected at comparable locations and operations in the various plants are tabulated in tables 8, 9, 10 and 11. Samples which were not comparable were not included in these tables, but were utilized in the calculation of weighted average exposures for the specific occupations at each plant.

Table 8 shows the frequency distribution of air concentrations of water soluble, hexavalent chromium according to the eight broad functional groupings in the chromate-producing industry. It will be noted that hexavalent chromium exists throughout the various operations in the industry. However, by comparison, air concentrations associated with ore processing are low. "Roasting" operations provided air concentrations of the greatest magnitude.

Table 9 shows the frequency distribution of air concentrations of acid soluble-water insoluble chromium according to the eight functional groups. It is evident that the basic chromium sulfate associated with tanning compound manufacture produced high air concentrations in areas of operation related to the group "other products" and also affected the group "filtering, drying and packing." Acid soluble-water insoluble chromium originating from the roast and residues produced moderately heavy concentrations throughout the dry end and extended into the wet end, as shown by the presence of some high concentrations found associated with "neutralizing and treating."

Table 10 shows the frequency distribution of air concentrations of chromite ore for the eight functional groups. These data show that very high air concentrations were associated with ore processing operations and relatively low ones with the other subsequent process groups, especially the wet-end functions "concentrating and granulating," and "filtering, drying and packing." Concentrations of chromite ore were also very low in air samples associated with the production of other products.

Table 11 shows the frequency distribution of air concentrations of total chromium for the eight functional groups, which represents a summation of tables 8, 9, and 10.

### Particle size determination *(112)*

A limited study was made of the particle size distribution of the airborne dust in a chromate-producing plant. Two simultaneous thermal precipitator

TABLE 8.—*Frequency distribution of air concentrations of hexavalent chromium for functional groups from all plants.*

| Functional group | Total number of samples | Percent of samples within indicated range (milligrams Cr per cubic meter of air) | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | 0.00 0.05 | 0.06 0.10 | 0.11 0.15 | 0.16 0.20 | 0.21 0.25 | 0.26 0.30 | 0.31 0.35 | 0.36 0.40 | 0.41 0.50 | 0.51 0.60 | 0.61 0.70 | 0.71 0.80 | 0.81 0.90 | 0.91 1.0 | 1.01 1.50 | 1.51 2.00 | 2.01 3.00 | over 3.00 |
| Ore processing | 76 | 92 | 7 | 1 | ...... | ...... | ...... | ...... | ...... | ...... | ...... | ...... | ...... | ...... | ...... | ...... | ...... | ...... | ...... |
| Residue processing | 90 | 51 | 18 | 10 | 2 | 6 | 2 | 4 | 4 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | ...... | ...... |
| Roasting | 116 | 53 | 14 | 9 | 4 | 3 | 2 | 2 | 2 | 2 | 0 | 1 | 1 | 1 | 1 | 2 | 0 | 2 | 1 |
| Leaching | 98 | 38 | 22 | 7 | 11 | 6 | 5 | 1 | 2 | 3 | 2 | 1 | 0 | 1 | 0 | 0 | 0 | 1 | ...... |
| Neutralizing and treating | 206 | 51 | 15 | 14 | 5 | 5 | 1 | 2 | 1 | 2 | 1 | 1 | ½ | 1 | ½ | ...... | ...... | ...... | ...... |
| Concentrating and granulating | 84 | 63 | 25 | 7 | 2 | 1 | 0 | 2 | ...... | ...... | ...... | ...... | ...... | ...... | ...... | ...... | ...... | ...... | ...... |
| Filtering, drying and packing | 101 | 46 | 11 | 5 | 6 | 9 | 4 | 1 | 2 | 4 | 2 | 4 | 4 | 0 | 1 | 0 | 0 | 0 | 1 |
| Other products | 169 | 68 | 16 | 5 | 2 | 3 | 1 | 1 | 1 | ½ | 0 | ½ | 0 | ½ | 1 | 0 | 0 | 0 | ½ |

TABLE 9.—*Frequency distribution of air concentrations of acid soluble-water insoluble chromium for functional groups from all plants.*

| Functional group | Total number of samples | Percent of samples within indicated range (milligrams Cr per cubic meter of air) | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | 0.00 0.05 | 0.06 0.10 | 0.11 0.15 | 0.16 0.20 | 0.21 0.25 | 0.26 0.30 | 0.31 0.35 | 0.36 0.40 | 0.41 0.50 | 0.51 0.60 | 0.61 0.70 | 0.71 0.80 | 0.81 0.90 | 0.91 1.0 | 1.01 1.50 | 1.51 2.00 | 2.01 3.00 | over 3.00 |
| Ore processing | 28 | 54 | 18 | 11 | 10 | 0 | 4 | 0 | 3 | ...... | ...... | ...... | ...... | ...... | ...... | ...... | ...... | ...... | ...... |
| Residue processing | 16 | 25 | 38 | 7 | 12 | 0 | 6 | 0 | 6 | 6 | ...... | ...... | ...... | ...... | ...... | ...... | ...... | ...... | ...... |
| Roasting | 32 | 69 | 13 | 3 | 9 | 0 | 3 | 0 | 0 | 3 | ...... | ...... | ...... | ...... | ...... | ...... | ...... | ...... | ...... |
| Leaching | 21 | 62 | 0 | 0 | 14 | 14 | 0 | 0 | 0 | 10 | ...... | ...... | ...... | ...... | ...... | ...... | ...... | ...... | ...... |
| Neutralizing and treating | 32 | 69 | 9 | 4 | 6 | 3 | 0 | 0 | 0 | 6 | 0 | 0 | 0 | 0 | 0 | 3 | ...... | ...... | ...... |
| Concentrating and granulating | 10 | 80 | 20 | ...... | ...... | ...... | ...... | ...... | ...... | ...... | ...... | ...... | ...... | ...... | ...... | ...... | ...... | ...... | ...... |
| Filtering, drying and packing | 15 | 67 | 7[1] | 7[1] | 0 | 7[1] | 0 | 6[1] | 0 | 0 | 0 | 0 | 6[1] | ...... | ...... | ...... | ...... | ...... | ...... |
| Other products | 30 | 53 | 7 | 10[1] | 4[1] | 3 | 0 | 0 | 0 | 0 | 6[1] | 0 | 0 | 0 | 0 | 3[1] | 3[1] | 4[1] | 7[1] |

1 Primarily tanning compound; Cr as basic chromium sulfate.

TABLE 10.—*Frequency distribution of air concentrations of chromite ore for functional groups from all plants.*

| Functional group | Total number of samples | Percent of samples within indicated range (milligrams Cr per cubic meter of air) | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | 0.00 0.05 | 0.06 0.10 | 0.11 0.15 | 0.16 0.20 | 0.21 0.25 | 0.26 0.30 | 0.31 0.35 | 0.36 0.40 | 0.41 0.50 | 0.51 0.60 | 0.61 0.70 | 0.71 0.80 | 0.81 0.90 | 0.91 1.0 | 1.01 1.50 | 1.51 2.00 | 2.01 3.00 | over 3.00 |
| Ore processing | 28 | 25 | 11 | 7 | 4 | 0 | 4 | 0 | 3 | 0 | 7 | 0 | 0 | 3 | 0 | 18 | 7 | 7 | 4 |
| Residue processing | 16 | 50 | 25 | 13 | 0 | 0 | 0 | 0 | 12 | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... |
| Roasting | 32 | 59 | 19 | 4 | 0 | 6 | 3 | 0 | 3 | 0 | 0 | 0 | 3 | 3 | ..... | ..... | ..... | ..... | ..... |
| Leaching | 21 | 70 | 0 | 0 | 10 | 5 | 0 | 0 | 0 | 5 | 0 | 5 | 0 | 0 | 0 | 5 | ..... | ..... | ..... |
| Neutralizing and treating | 33 | 76 | 15 | 3 | 3 | 3 | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... |
| Concentrating and granulating | 10 | 100 | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... |
| Filtering, drying and packing | 15 | 100 | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... |
| Other products | 31 | 97 | 3 | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... |

TABLE 11.—*Frequency distribution of air concentrations of total chromium for functional groups from all plants.*

| Functional group | Total number of samples | Percent of samples within indicated range (milligrams Cr per cubic meter of air) | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | 0.00 0.05 | 0.06 0.10 | 0.11 0.15 | 0.16 0.20 | 0.21 0.25 | 0.26 0.30 | 0.31 0.35 | 0.36 0.40 | 0.41 0.50 | 0.51 0.60 | 0.61 0.70 | 0.71 0.80 | 0.81 0.90 | 0.91 1.0 | 1.01 1.50 | 1.51 2.00 | 2.01 3.00 | over 3.00 |
| Ore processing | 28 | 14 | 7 | 18 | 4 | 0 | 7 | 3 | 0 | 4 | 0 | 4 | 0 | 4 | 0 | 14 | 11 | 7 | 3 |
| Residue processing | 16 | 6 | 6 | 12 | 6 | 6 | 13 | 13 | 7 | 19 | 0 | 0 | 6 | 0 | 0 | 6 | ..... | ..... | ..... |
| Roasting | 32 | 6 | 13 | 13 | 16 | 3 | 9 | 6 | 0 | 12 | 4 | 6 | 3 | 0 | 0 | 3 | 3 | 0 | 3 |
| Leaching | 15 | 6 | 13 | 13 | 7 | 0 | 7 | 7 | 0 | 13 | 0 | 7 | 0 | 0 | 0 | 7 | 7 | 7 | 6 |
| Neutralizing and treating | 33 | 9 | 15 | 12 | 9 | 9 | 9 | 7 | 6 | 6 | 6 | 0 | 0 | 3 | 3 | 0 | 3 | 3 | ..... |
| Concentrating and granulating | 10 | 30 | 10 | 20 | 30 | 0 | 0 | 10 | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... |
| Filtering, drying and packing | 15 | 0 | 0 | 27 | 7 | 13 | 7 | 7 | 0 | 7 | 13 | 7 | 0 | 0 | 0 | 6 | 0 | 0 | 6 |
| Other products | 30 | 27 | 13 | 17 | 13 | 4 | 4 | 0 | 0 | 0 | 3 | 0 | 0 | 3 | 0 | 3 | 0 | 3 | 10 |

samples were collected in the roasting and leaching area of a chromate plant and used for these particle size determinations. One-hour samples

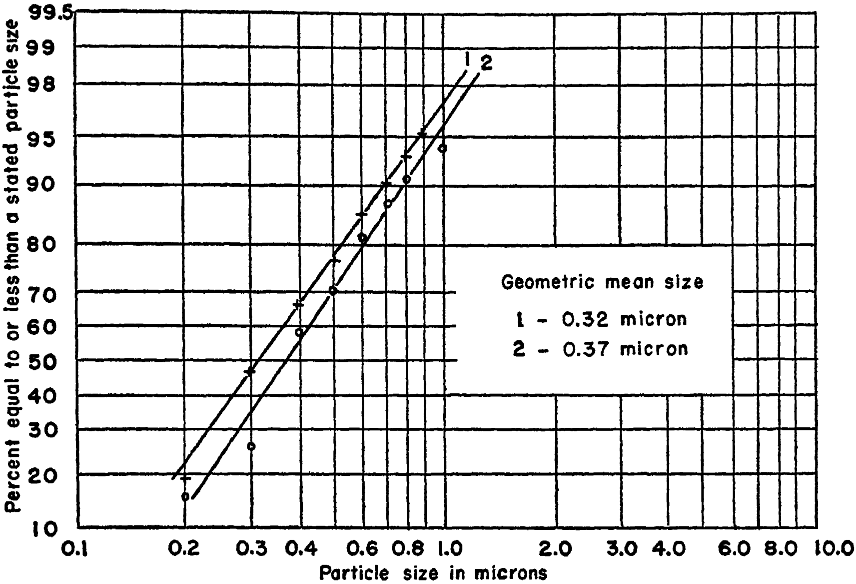


**FIGURE 4.—Particle size distributions of two simultaneous samples of airborne dust collected with the thermal precipitator in roasting and leaching area of a chromate-producing plant.**

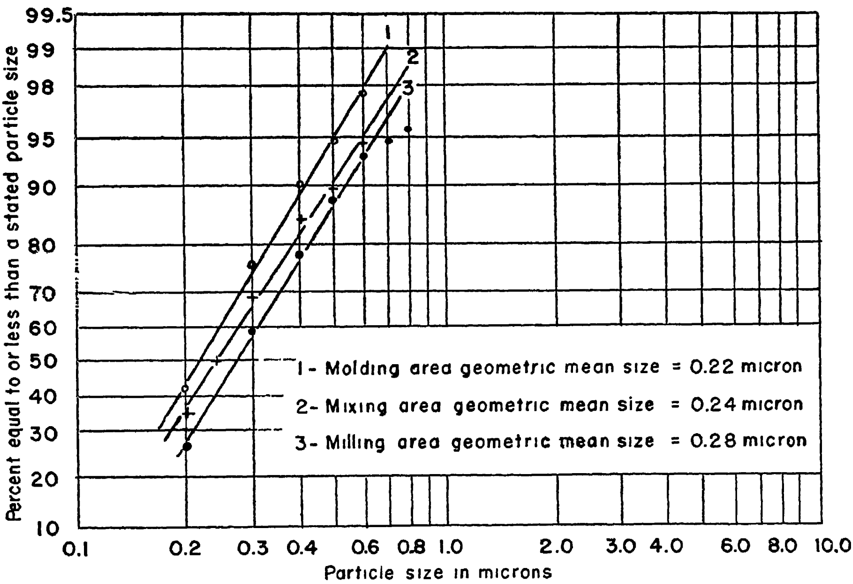


**FIGURE 5.—Particle size distribution of airborne dusts from comparison plant C collected with the thermal precipitator.**

were collected at a rate of 6.0 ml per minute. The size distributions are plotted in Figure 4. The geometric mean particle size averaged 0.35 micron and the standard geometric deviation was 0.18.

Also a comparison study was made of the particle size distributions of the airborne dusts in the three main working areas of comparison plant C, which makes refractory brick using a chromite ore. Figure 5 represents the particle size distributions of airborne dusts in working areas 1, 2, and 3. Forty-five minute thermal precipitator samples were used for these particle size determinations.

TABLE 12.—*Average dust counts of samples taken from two comparison plants and one chromate-producing plant.*

| Plant | Average dust count (million particles per cubic foot of air) |
|---|---|
| *Comparison Plant A*—(Zeiss konimeter samples) | |
| Gel plant K1 | 21 |
| Gel plant K2-K10, first level | 35 |
| Gel plant K2-K10, second level | 27 |
| Shops | 10 |
| *Comparison Plant C*—(Standard impinger samples) | |
| Ore milling area | 270 |
| Mixing area | 71 |
| Molding room | 41 |
| *Chromate-producing plant*— (Zeiss konimeter samples) | |
| Ore milling area | 16 |
| Kiln area | 17 |
| Leach area | 14 |
| Residue mill area | 9 |
| Residue mix area | 8 |

TABLE 13.—*Weighted averages of exposures to water soluble, hexavalent chromium, by occupational group.*

| Occupational group | Hexavalent chromium (water soluble) | | | |
|---|---|---|---|---|
| | Range of means $CrO_3$ [1] | Mean $CrO_3$ [1] | Mean Cr [2] | Principal exposure |
| Ore processors | 0.00-0.08 | 0.02 | 0.01 | Chromate. |
| Lime and soda ash handlers | 0.00-0.08 | .02 | .01 | Do. |
| Mill room laborers | .01-.16 | .08 | .04 | Do. |
| Mix operators (ore) | .00-.10 | .04 | .02 | Do. |
| Mix operators (residue) | .25-.28 | .27 | .14 | Do. |
| Kiln operators | .01-.62 | .17 | .09 | Do. |
| Kiln building laborers | .01-.28 | .15 | .08 | Do. |
| Crane operators | .08-.57 | .28 | .14 | Do. |
| Leach operators | .06-.53 | .22 | .11 | Do. |
| Mud operators | .05-.16 | .09 | .05 | Do. |
| Residue drier operators | .01-.31 | .13 | .07 | Do. |
| Residue mill operators | .04-.22 | .14 | .07 | Do. |
| Alumina recovery operators | .01-.23 | .12 | .06 | Do |
| Evaporator operators | .02-.06 | .04 | .02 | Bichromate. |
| Chemical treat operators | .02-.22 | .14 | .07 | Do. |
| Sulfate recovery operators | .01-.52 | .23 | .12 | Do. |
| Liquor concentration operators | .08-.08 | .08 | .04 | Do. |
| Centrifuge operators | .02-.43 | .27 | 14 | Do |
| Drying and bagging operators | .03-.50 | .17 | .09 | Do. |
| Shippers | .01- 06 | .03 | .02 | Do. |
| Chromic acid cookers and packers | .04-.08 | .06 | .03 | Do. |
| Potash production operators | .04-.83 | .33 | .17 | Do. |
| Chromate operators | .03-.12 | .08 | .04 | Chromate. |
| Tanning compound operators | .01-.02 | .01 | .005 | Bichromate. |

[1] Milligrams $CrO_3$ per cubic meter of air.
[2] Milligrams Cr per cubic meter of air.

Figures 4 and 5 show that the mean sizes of the airborne dusts in these two plants are small compared to industrial dusts as generally reported; namely, about 0.25 micron compared to about one micron, and that the airborne dusts in these two industries are comparable in size distribution.

TABLE 14.—*Weighted averages of exposures to acid soluble-water insoluble chromium, by occupational group.*

| Occupational group | Acid soluble-water insoluble chromium | | |
|---|---|---|---|
| | Range of means (milligrams Cr per cubic meter of air) | Mean (milligrams Cr per cubic meter of air) | Principal exposure |
| Ore processors | 0.00-0.17 | 0.05 | Residue. |
| Lime and soda ash handlers | .01-.08 | .03 | Do. |
| Mill room laborers | .05-.22 | .14 | Do. |
| Mix operators (ore) | .0-.18 | .06 | Do. |
| Mix operators (residue) | .12-.22 | .17 | Do. |
| Kiln operators | .01-.14 | .06 | Roast and residue. |
| Kiln building laborers | 0-.12 | .07 | Do. |
| Crane operators | .01-.21 | .07 | Do. |
| Leach operators | .01-.08 | .04 | Do. |
| Mud operators | 0-.03 | .01 | Residue. |
| Residue drier operators | 0-.15 | .06 | Do. |
| Residue mill operators | .11-.18 | .15 | Do. |
| Alumina recovery operators | 0-.10 | .04 | Unknown. |
| Evaporator operators | 0-.02 | .01 | Do. |
| Chemical treat operators | .01-.21 | .05 | Do. |
| Sulfate recovery operators | 0-.18 | .08 | Do. |
| Liquor concentration operators | 0-.04 | .02 | Basic chromium sulfate |
| Centrifuge operators | 0-.10 | .04 | Do. |
| Drying and bagging operators | 0-.06 | .02 | Do. |
| Shippers | .01-.30 | .15 | Do. |
| Chromic acid cookers and packers | .02-.02 | .02 | Unknown. |
| Potash production operators | 0-.01 | 0 | Do. |
| Chromate operators | 0- 0 | 0 | Do. |
| Tanning compound operators | .34-.60 | .47 | Basic chromiun sulfate. |

TABLE 15.—*Weighted averages of exposures to chromite ore, by occupational group.*

| Occupational group | Chromite ore | | |
|---|---|---|---|
| | Range of plant means (milligrams $Cr_2O_3$ per cubic meter of air) | Mean (milligrams $Cr_2O_3$ per cubic meter of air) | Mean (milligrams Cr per cubic meter of air) |
| Ore processors | 0.05- 1.6 | 0.53 | 0.36 |
| Lime and soda ash handlers | .05- .60 | .25 | .17 |
| Mill room laborers | .65- 2.0 | 1.3 | .89 |
| Mix operators (ore) | .07- 2.5 | 1.0 | .69 |
| Mix operators (residue) | .05- .18 | .12 | .08 |
| Kiln operators | 0- .28 | .07 | .05 |
| Kiln building laborers | .02- .33 | .10 | .07 |
| Crane operators | .02- .17 | .07 | .05 |
| Leach operators | .01- .12 | .06 | .04 |
| Mud operators | 0- .03 | .01 | .007 |
| Residue drier operators | .02- .08 | .05 | .03 |
| Residue mill operators | .04- .55 | .25 | .17 |
| Alumina recovery operators | 0- .10 | .05 | .03 |
| Evaporator operators | .01- .02 | .01 | .007 |
| Chemical treat operators | 0- .05 | .02 | .01 |
| Sulfate recovery operators | .03- .08 | .04 | .03 |
| Liquor concentration operators | 0- .01 | 0 | 0 |
| Centrifuge operators | 0- .07 | .03 | .02 |
| Drying and bagging operators | 0- .02 | .01 | .007 |
| Shippers | 0- .01 | 0 | 0 |
| Chromic acid cookers and packers | 0- .01 | 0 | 0 |
| Potash production operators | 0- .01 | 0 | 0 |
| Chromate operators | 0- .01 | 0 | 0 |
| Tanning compound operators | 0- 0 | 0 | 0 |

### Average dust counts

Table 12 shows the dust concentrations in working areas of two comparison plants and one chromate-producing plant. These data indicate that the working environment of both comparison plants A and C are equally as dusty as, or dustier than, that of the chromate-producing plant.

## ***Weighted Exposures***

Weighted exposures are shown in tables 13, 14, and 15 for the production operations. These data were obtained by weighting the average concentration of an air contaminant found in a certain area according to the time a given workman spends in that area. For example, if, at a certain plant in a given job, the workman spends five hours a day at task A, where there is an average dust concentration of 0.60 mg $CrO_3$ per cubic meter; two hours a day in area B, where there is a dust concentration of 0.94 mg $CrO_3$ per cubic meter; and one hour a day at task C, where the dust concentration is 0.08 mg $CrO_3$ per cubic meter, the weighted exposure for this occupation in this plant would be:

$$\frac{(5 \times 0.60) + (2 \times 0.94) + (1 \times 0.08)}{8} = 0.62 \text{ mg } CrO_3/m^3$$

In Figure 6 the mean weighted average exposures to total chromium for the industry-wide job classifications are presented. The total chromium is broken down into the three determined forms of chromium. The black portions of the diagram represent those fractions of the total chromium which existed as chromite ore. The cross-hatched portions of the diagrams represent those fractions of the total chromium which existed as acid soluble-water insoluble chromium. The clear portions of the diagrams represent those fractions of the total chromium which existed as water soluble hexavalent chromium. The dotted cross-hatched portion of the diagram represents those fractions of the total chromium which existed as acid soluble-water insoluble chromium, but whose origin is known to be mainly basic chromium sulfate tanning compounds.

Figure 6 shows that in the average plant the greatest average exposure to chromium-bearing dusts occurs in the dry end with the maximum mean exposure occurring for the occupational group "mill room laborers." This amounts to an exposure of 1.07 milligrams of total chromium per cubic meter of air. Eighty-three percent (0.89 mg $Cr/m^3$) of this exposure is due to chromium in the form of chromite ore, 13 percent (0.14 mg $Cr/m^3$) to chromium in the form of acid soluble-water insoluble chromium, and 4 percent (0.04 mg $Cr/m^3$) to water soluble, hexavalent chromium.

Also, this figure shows that the greatest average exposure (0.17 mg $Cr/m^3$) to hexavalent chromium occurs for the occupational group "potash production operators." However, several other occupational groups which are associated with various phases of the process approach this level of exposure. For example, the following occupations also have average exposures

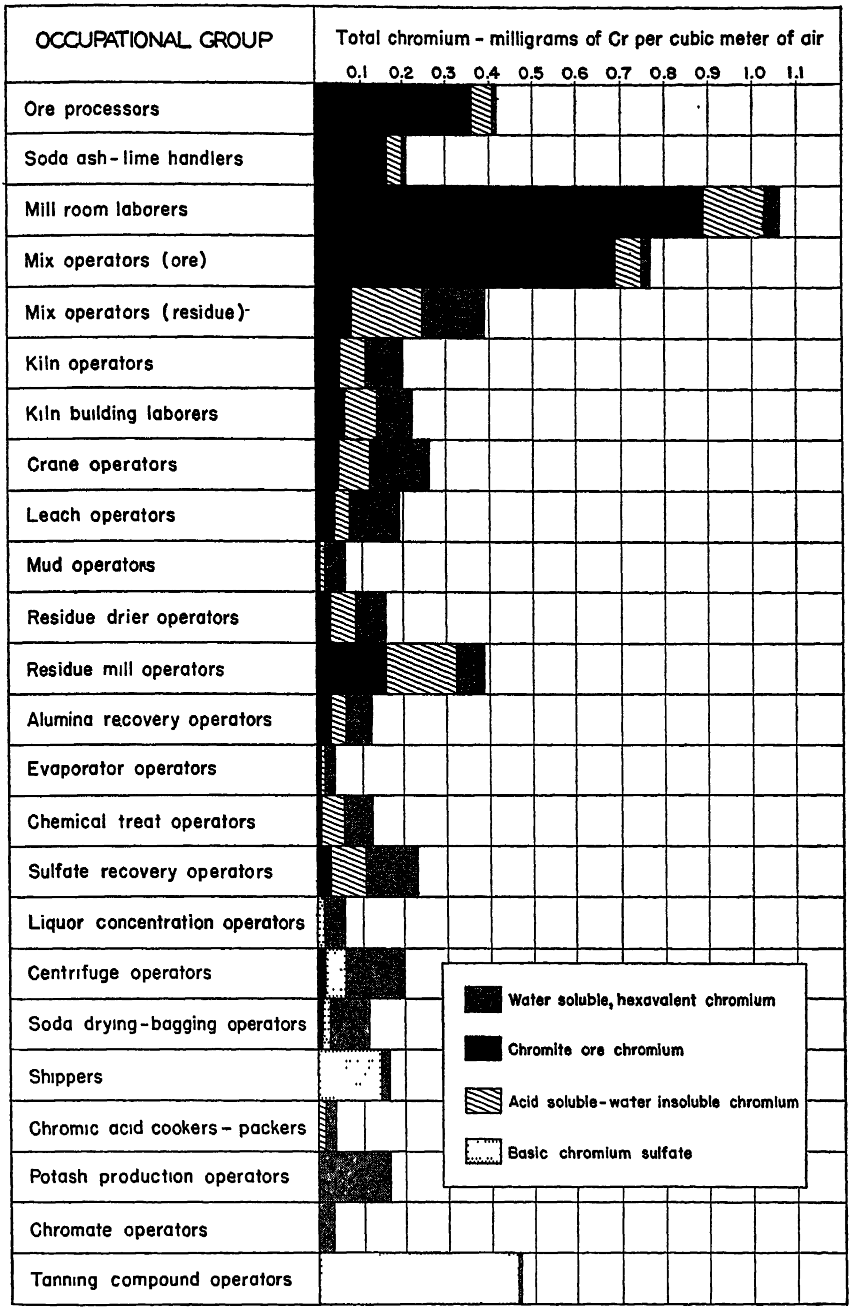


**FIGURE 6.—Mean weighted averages of exposures for all chromate-producing plants.**

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greater than 0.10 mg of water soluble, hexavalent chromium per cubic meter of air: residue mix operators, crane operators, leach operators, sulfate recovery operators, and centrifuge operators.

The two occupational groups which process residue have the greatest average exposure to acid soluble-water insoluble chromium. These occupational groups are the residue mix operators (0.17 mg $Cr/m^3$) and residue mill operators (0.15 mg $Cr/m^3$). All other dry-end occupations and some of the initial wet-end occupations have some exposure to this form of chromium.

## Dust Control Measures

Many of the operations in this industry produce a large amount of dust. However, all of the contributing sources could be controlled and the degree of dustiness which was observed in the industry could be diminished markedly by use of established industrial hygiene engineering principles. Chiefly, the enclosure of dusty operations could be used to especially good advantage in many instances. Better process enclosures and tighter material handling systems, provided with exhaust systems which maintain slightly negative pressures at all times within these enclosures, would eliminate almost all of the sources of air contamination within the plants.

It must be mentioned at this point that maintenance of these tighter systems together with maintenance of efficient exhaust system ventilation is equally as important as the provision of these control measures. One of the highest exposure levels in the industry was found in a building where control features were provided, but operating inefficiently.

Also, housekeeping is an extremely important factor in the maintenance of good dust control. For example, at one plant where the operators hosed down their working areas with water each shift, the plant average exposure for all operations was 0.02 mg of total chromium per cubic meter of air. At another plant where this same degree of housekeeping was obviously lacking and the application of dust control measures was also less efficient or lacking, the plant average exposure was 0.17 mg of total chromium per cubic meter of air.

Several major sources of air contamination observed in the industry are worthy of note. They are listed below with a discussion and suggested means of control as shown in figure 7.

### Roast crushing

Two plants crush the roast prior to leaching. The dustiness of these operations was obvious and was borne out by the results of the air samples. As much as 6.9 mg $CrO_3$ was found per cubic meter of air in the general area of this source. Complete enclosure with slight negative pressure within the enclosure would prevent the dissipation of this dust to other locations within the plant.

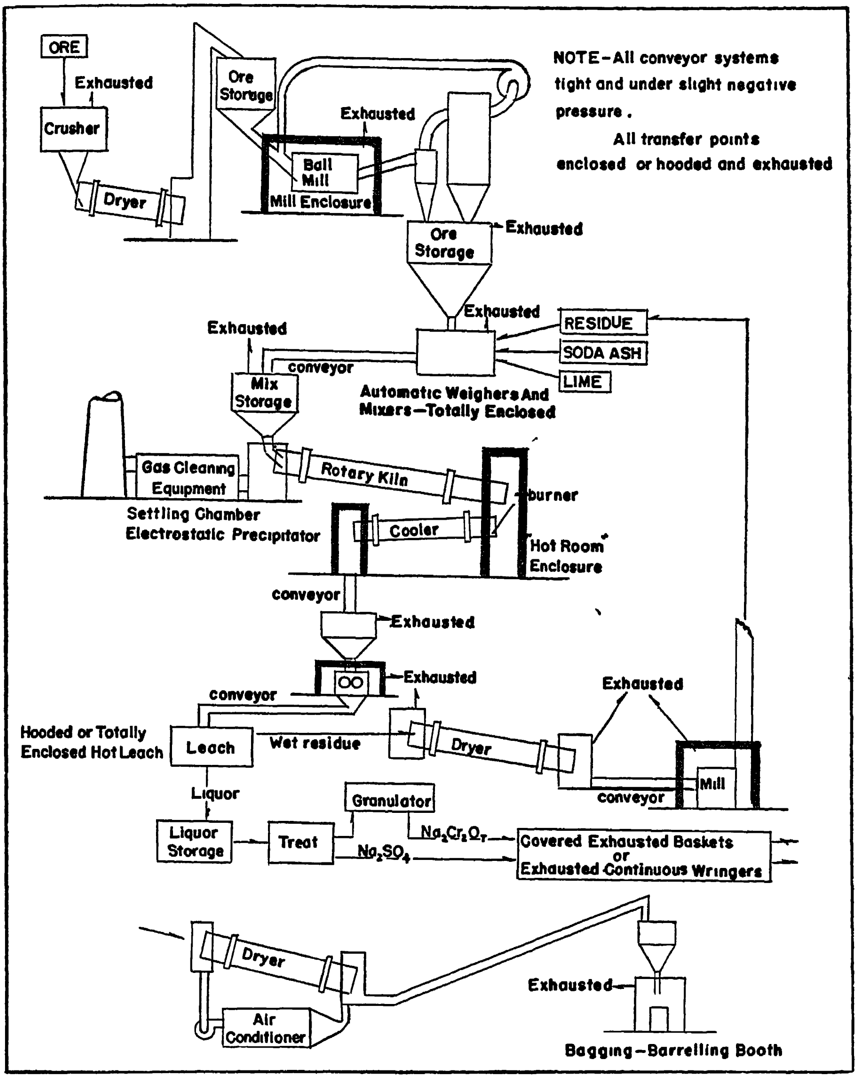


**FIGURE 7.**—Composite process diagram incorporating proved control features from various chromate-producing plants.

## Roasting

At each end of the rotary kilns and at each end of the rotary coolers (if used) is a material transfer point and, therefore, a potential source of dust dissemination. One plant has solved the problem of controlling roast dust at the discharge ends of the kilns by enclosing the discharge ends in a walled-off enclosure referred to as the "hot room." This enclosure also houses a chain conveyor which conveys the roast to an enclosed and exhausted conveyor system. The kilns are operated from the outside of the enclosure. The average air concentration in the kiln burner area at this

plant was 0.006 mg $CrO_3$ per cubic meter of air. This may be contrasted with another set of kilns without this enclosure which averaged 2.8 mg $CrO_3$ per cubic meter of air in the kiln burner area.

### Conveying

Several types of dry material conveyors were observed to be satisfactory so long as the control features were utilized. In order to maintain low air concentration of dust in the vicinity of conveying equipment, the system had to be absolutely tight or always under negative pressure, and each transfer point had to be enclosed and exhausted. Tightness is necessary not only to prevent dust dissemination, but to prevent gross spillage. Oversize conveyors will provide an airspace which allows air to flow and, therefore, it is possible to maintain a negative pressure in such a system.

The magnitude of the dust sources which may originate in conveying equipment may be appreciated from the following data:

An air sample collected two feet from an opening in a roast conveyor showed 1,820 mg $CrO_3$ per cubic meter of air. This particular system was neither tight nor exhausted.

### Leaching

The leach process has two phases which contribute significantly to the contamination of the plant atmosphere. The first major dust source is the transfer of roast to the leach filter boxes or leaching tanks. Where the material is allowed to fall from a conveyor into open boxes, the air concentration in the general area was found to be 3.2 mg $CrO_3$ per cubic meter of air.

The second major dust source is produced by the addition of leach liquor to hot roast. This procedure produces a boiling liquor which emits large volumes of steam. An air sample taken in the general area adjacent to a hot leach tank showed a concentration of 2.5 mg $CrO_3$ per cubic meter of air. A stack on a leach mill which terminated in the work room emitted steam which contained 148 mg $CrO_3$ per cubic meter of air.

### Liquor handling

Liquor conveying lines in this industry contribute a large proportion of the spillage in the wet end. Many pipe fittings and valves were observed to leak, and ceilings, walls and floors were often covered with the moist crystalline chromates or bichromates.

Air concentrations were found to be high in areas surrounding open liquor tanks where liquor was being agitated or was spilling into the tank. An air sample taken adjacent to an open floor well with liquor flowing into it showed 1.3 mg $CrO_3$ per cubic meter of air. A sample taken adjacent to an open liquor tank being filled with liquor and being agitated with a steam line showed an air concentration of 3.2 mg $CrO_3$ per cubic meter of air. A sample taken in the area adjacent to a partially open liquor receiving tank where liquor and wash water were received from a continuous centrifuge, showed an air concentration of 20 mg $CrO_3$ per cubic meter of air.

A large leak which developed in a liquor line under pressure sprayed liquor throughout a particular work room. Five minutes after the flow was stopped and production was resumed, the general air concentration was 1.5 mg $CrO_3$ per cubic meter of air.

Better piping design and better maintenance are needed to prevent the gross spillage by the liquor conveying system. Also more frequent cleaning is needed to maintain clean work rooms.

Liquor receiving tanks, as well as all other transfer points, should be enclosed more completely and exhausted.

### Filtering and centrifuging

The spray emitted by a bichromate wringer with no exhaust or enclosure averaged 7.7 mg $CrO_3$ per cubic meter of air. An air sample taken immediately adjacent to a sulfate wringer showed an air concentration of 1.8 mg $CrO_3$ per cubic meter of air.

Air concentrations in the vicinity of continuous centrifuges were lower than those near open basket-type centrifuges. Complete enclosure of the wringer with this enclosure exhausted gave the best control. A window in the enclosure allowed the operator to see the basket being emptied by a screw-feed scraper. Exposure levels averaged 0.018 mg $CrO_3$ per cubic meter of air for this controlled-type of operation, as contrasted with an average exposure level of 0.23 mg $CrO_3$ per cubic meter of air for an open basket with no exhaust.

### Sulfate handling

Sodium sulfate is a voluminous product of the industry and is disposed of without purification in all but one instance. This salt, if pure, would be of minor importance in this discussion, but since it contains a small percentage of chromium, the large amounts of sulfate dust produced in some instances in the drying, storage, and general handling procedures provide a major source of soluble chromium air contamination. In the immediate vicinity of a sulfate drier without dust control equipment, for example, air concentrations were found to be 19 mg sodium sulfate per cubic meter and 0.30 mg $CrO_3$ per cubic meter. This dust so permeated the area that air concentrations as high as 4.5 mg sodium sulfate per cubic meter were found in remote parts of the same building.

Large amounts of sulfate were observed to have settled out of the air in the vicinity of these operations and also in many adjacent buildings. Settled dust from one kiln burner area contained a large percentage of sodium sulfate dust which originated at a conveyor system that filled railroad cars with the salt at a location adjacent to the kiln building. Dust and mist control facilities should be incorporated with the sulfate wringers, driers, and dry sulfate conveyors as with the corresponding equipment handling the bichromate.

### Bagging and barreling

Bagging, barreling and packaging are best done in an exhausted booth. A local exhaust hood about the perimeter of the hopper feed pipe gives good results in most cases. However, in one instance, where the feed tank was under slight positive pressure, an air sample taken at the bagging operation showed an air concentration of 420 mg $CrO_3$ per cubic meter when the feed tank emptied and the dust laden air escaped from the tank. Packing is an intermittent procedure which continues until the feed hopper is emptied and then resumes when the supply in the feed hopper is replenished. In all instances, therefore, it is good practice to maintain a slight negative pressure in this feed tank. This also prevents escape of dust if there are openings in the feed tank. An air sample taken in the vicinity of a two-square-foot opening at the top of a bichromate storage hopper (without exhaust) contained 12 mg $CrO_3$ per cubic meter. Also, an exhausted shaker screen, with a large cover purposely left wide open to facilitate operation of the screen, produced air concentrations in that area of 2 mg $CrO_3$ per cubic meter.

Good enclosure should be maintained on these systems, and these enclosures should be exhausted. Where work procedures demand openings in the system, the exhaust system should be designed to allow for these openings and provide velocities through these openings sufficient to give the necessary control.

## *Summary and Recommendations*

### Summary

A description is given of the basic processes involved in the manufacture of chromates and bichromates from chromite ore. Flow diagrams show the relationships of the various operations.

Approximately 1,800 samples were collected throughout the industry for the purpose of defining the atmospheric environment. The great majority of these were air samples, but material and settled dust samples were also collected.

It was found that the dry-end processes generate dusts containing chromite ore, soda ash, roast, residue, and sodium chromate. Sodium bichromate and sodium sulfate are usually found associated only with the wet-end processes. Wet-end processes are associated with acid solutions while dry-end processes are associated with alkaline materials and solutions.

An appreciable portion of the total chromium is present in an acid soluble-water insoluble state, indicating the presence of a form or forms of chromium which are dissimilar from either insoluble chromite ore or water soluble, hexavalent chromium. Roasting, leaching, neutralizing and treating operations have the largest proportion of acid soluble-water insoluble chromium. However, settled dust samples from many areas not associated with roast or

residue processes had high percentages of acid soluble-water insoluble chromium.

A limited study of the particle size distribution of the airborne dust in a chromate-producing plant showed a geometric mean of 0.35 micron and a standard deviation of 0.18.

The greatest weighted average exposure to chromium-bearing dusts occurs in the dry end with the maximum mean exposure of 1.07 milligrams of total chromium per cubic meter of air found for mill room laborers. The greatest weighted average exposure (0.17 mg $Cr/m^3$) to hexavalent chromium occurs among potash production operators. Occupations with average exposures greater than 0.10 mg of water soluble, hexavalent chromium per cubic meter of air include residue mix operators, crane operators, leach operators, sulfate recovery operators and centrifuge operators. The two occupational groups which process residue have the greatest average exposure to acid soluble-water insoluble chromium. These groups are residue mix operators (0.17 mg $Cr/m^3$) and residue mill operators (0.15 mg $Cr/m^3$).

## Recommendations

The problems involved in the control of the dusts in this industry are not unusual but are generally typical of the chemical industry and require the application of the established industrial hygiene engineering principles.

Facilities and competent personnel should be provided to perform routine air analyses throughout each of the plants in order to better control the various atmospheric contaminants and also to ascertain the current exposure levels. Data should be accurately recorded for correlation with any future medical findings.

Adequate dust control features should be incorporated in the design of all new equipment and in the redesign of old equipment in accordance with the discussion beginning on page 38. More complete enclosure of process equipment and conveying systems, as well as greater use of local exhaust ventilation, should be utilized to minimize the contamination of the plant air.

Housekeeping methods should be perfected to prevent accumulation of dusts and spillage in the plants. Wet cleaning of working areas is advisable, wherever possible.

Great effort should be made to provide air sufficiently clean to eliminate the general need for the use of respirators. However, until air concentrations are reduced to a safe level, or on special operations where this degree of control is not practicable, or where it is necessary for a workman to enter a control enclosure or area of breakdown, then it shall be required that personal protective devices, such as approved type dust respirators, be worn continuously during the period of exposure.

# MORBIDITY AND MORTALITY EXPERIENCE

Data were collected on paid death claims and cases of sickness and non-industrial injuries disabling for 8 calendar days or longer among members of the sick-benefit plans of the 7 plants. Periods for which records of these plans were available varied from two to eleven years. The rules and regulations of the plans may be summarized as follows:

In all of the plants, benefits began on the eighth day of disability. Prior to 1949 the maximum period for which benefits were paid was 13 weeks. With the passage of a sickness insurance law in New Jersey, effective on January 1, 1949, plants located in that state had maximum benefits extended to 26 weeks. However, in order that the data for all companies be comparable, cases lasting longer than 98 days were arbitrarily closed at 98 days. The calendar days of disability, therefore, are defined as the number of calendar days from the date of onset of disability to the date of return to work or to the 98th day, inclusive, except in the event the employee died or was pensioned before the 98th day.

Before 1949 the probationary period or waiting period between employment and eligibility for membership in the sickness insurance plan was 6 months for 2 plants, 3 months for 1 plant, and 1 month for 3 plants; 1 plant had no plan. After the New Jersey state sickness insurance law went into effect in January 1949 and the New York law became effective on July 1, 1950, workers in these areas were eligible for benefits as soon as they were employed. By the end of 1950 one plant still had a waiting period of 6 months, one plant had a waiting period of one month, and the remaining 5 plants, located in New York and New Jersey, had no probationary periods.

At first, membership was voluntary in all plants, but after the enactment of the New Jersey and New York laws, membership in the sickness insurance plans was compulsory for plant workers in those states. In the two plants where membership remained voluntary, coverage was approaching 100 percent of all employees toward the end of the study period.

Although a medical examination was not required before a worker could become a member of the sickness insurance plan, all plants required a medical examination before employment. There were no age limits on eligibility for membership. However, some plants which allowed 26 weeks' benefits for each different disease during a year permitted only a total of 26 weeks per year for all diseases for persons over 60 years of age.

From the foregoing rules and regulations it is evident that the only newly-employed workers included in the study were those who entered the New Jersey plants during 1949 and 1950 and the New York plant during the last 6 months of 1950. Neither the morbidity nor mortality experience of employees during the probationary period (whether they quit or remained with the plant) was included.

The disability analysis is based on only those cases that occurred while a worker was a member of the sickness insurance plan. All disabilities that ended between January 1, 1946, and December 31, 1950, are included, provided that they lasted for 8 calendar days or longer. Hence, disabilities that began in 1945 and ended in 1946 are included. The actual number of days for all cases ending during the study period are counted up to the 98th day,

inclusive. Industrial injuries and compensated cases of occupational disease are not included.

The mortality analysis is based on information from the records of the sick benefit associations which contained data on death benefits. Only deaths of workers who were members of a sick benefit association and died within one year after becoming disabled are included. Sudden deaths and deaths before the 8th day of disability are included; although these cases were not eligible for sickness benefits, they were eligible for death benefits. Deaths due to industrial injuries are not included. All deaths were classified by cause as given on the death certificate without additional verification.

## *Disability Experience, 1946-50*

During the 5-year period, 1946–50, for the seven chromate plants there was a total of 5,121 person-years of membership, of which 3,663 and 1,458 represented white and colored males, respectively. It will be observed from the accompanying table that the proportion of colored persons in the total membership declines rapidly after 55 years of age. In the age group 65 years and over there were 136 white males as compared with 9 colored.

| Color | All ages | Age group (years) | | | | |
|---|---|---|---|---|---|---|
| | | Under 35 | 35–44 | 45–54 | 55–64 | 65 and over |
| | Average number of person-years of membership | | | | | |
| White males | 3,663 | 1,014 | 1,015 | 803 | 695 | 136 |
| Colored males | 1,458 | 483 | 442 | 409 | 115 | 9 |

Six of the plants reported during the full 5-year period with a steady increase in the average membership from 1946 through 1949. The seventh, a small plant, reported only during the last two years, 1949 and 1950.

### Frequency by year ended

As may be observed from table 16, the average annual number of cases on account of sickness and nonindustrial injuries was 130.9 per 1,000 white males and 186.2 per 1,000 colored males in 1946, as compared with 109.8 and 124.6, respectively, in 1950. The rates for the broad cause groups are not shown in this table, but an examination of those rates reveals that the abnormally low rate for white males in 1947 (86.0) is attributable to a marked decrease in respiratory diseases for that year. Colored males also showed a decrease in the respiratory disease rate for 1947, but this was more than counterbalanced by an increase in nonrespiratory-nondigestive diseases. There was a slight upward trend in the rates for nonindustrial injuries, while the rates for digestive diseases and nonrespiratory-nondigestive diseases fluctuated, but not in one particular direction.

## Frequency according to broad cause group, by color and duration

Frequency rates for white and colored males under 55 years of age according to duration of case are presented in table 17 for the broad cause groups. For sickness and nonindustrial injuries the colored rate for each duration is more than one and one-half times the white rate for the corresponding duration. The excess in the colored rate over the white rate is

TABLE 16.—*Average annual number of cases per 1,000 males on account of sickness and nonindustrial injuries disabling for 8 consecutive calendar days or longer, by color and year; experience of male members of sick benefit organizations of seven chromate-producing plants, 1946–50, inclusive.*[1]

[The data include experience of six plants during 1946–50, and one plant during 1949–50]

| Year in which case ended | Annual number of cases per 1,000 males | | | Number of cases | | | Average number of person years of membership | | |
|---|---|---|---|---|---|---|---|---|---|
| | Total | White | Colored | Total | White | Colored | Total | White | Colored |
| 1946–50 | 128.3 | 116.3 | 158.4 | 657 | 426 | 231 | 5,121 | 3,663 | 1,458 |
| 1946 | 147.3 | 130.9 | 186.2 | 123 | 77 | 46 | 835 | 588 | 247 |
| 1947 | 120.7 | 86.0 | 200.0 | 115 | 57 | 58 | 953 | 663 | 290 |
| 1948 | 145.6 | 137.8 | 164.5 | 151 | 101 | 50 | 1,037 | 733 | 304 |
| 1949 | 119.6 | 117.6 | 125.0 | 141 | 101 | 40 | 1,179 | 859 | 320 |
| 1950 | 113.7 | 109.8 | 124.6 | 127 | 90 | 37 | 1,117 | 820 | 297 |

1 Industrial injuries and venereal diseases are not included.

TABLE 17.—*Average annual number of cases per 1,000 males under 55 years of age on account of sickness and nonindustrial injuries disabling for 8 consecutive calendar days or longer, according to broad cause group, by color and duration; experience of male members of sick benefit organizations of seven chromate-producing plants, 1946–50, inclusive.*[1]

[The data include experience of six plants during 1946–50, and one plant during 1949–50]

| Duration of case in calendar days | Annual number of cases per 1,000 males under 55 years of age | | | | | |
|---|---|---|---|---|---|---|
| | White | Colored | White | Colored | White | Colored |
| | Sickness and nonindustrial injuries | | Nonindustria injuries | | Total sickness | |
| 8 days or longer | 86.2 | 156.7 | 8.1 | 17.3 | 78.1 | 139.4 |
| 15 days or longer | 68.5 | 121.4 | 5.6 | 15.0 | 62.9 | 106.4 |
| 22 days or longer | 45.2 | 86.2 | 3.2 | 8.2 | 42.0 | 78.0 |
| 29 days or longer | 34.3 | 60.7 | 2.5 | 5.2 | 31.8 | 55.5 |
| 43 days or longer | 23.3 | 35.2 | 1.8 | 2.2 | 21.5 | 33.0 |
| 57 days or longer | 15.9 | 29.2 | 1.1 | 1.5 | 14.8 | 27.7 |
| 92 days or longer | 10.2 | 15.7 | .3 | 0 | 9.9 | 15.7 |
| | Respiratory diseases | | Digestive diseases | | Nonrespiratory–nondigestive–diseases[2] | |
| 8 days or longer | 34.3 | 79.5 | 15.9 | 26.2 | 27.9 | 33.7 |
| 15 days or longer | 25.4 | 63.0 | 13.8 | 20.2 | 23.7 | 23.2 |
| 22 days or longer | 10.9 | 45.0 | 11.3 | 16.5 | 19.8 | 16.5 |
| 29 days or longer | 7.1 | 26.2 | 8.5 | 16.5 | 16.2 | 12.8 |
| 43 days or longer | 4.6 | 12.8 | 5.3 | 11.2 | 11.6 | 9.0 |
| 57 days or longer | 3.9 | 10.5 | 2.8 | 9.0 | 8.1 | 8.2 |
| 92 days or longer | 2.8 | 6.8 | 1.4 | 2.2 | 5.7 | 6.7 |
| Average number of person-years of membership | 2,832 | 1,334 | 2,832 | 1,334 | 2,832 | 1,334 |

1 Industrial injuries and venereal diseases are not included.
2 Ill-defined and unknown causes are included.

greatest for the respiratory diseases, next for the digestive diseases, and lowest for the nonrespiratory-nondigestive diseases. These differences can not be attributed to difference in age since the two colors have approximately the same age distribution.

## Frequency, disability, and severity rates according to detailed cause by color

The annual number of cases per 1,000 males, the annual number of days per male, and the average number of days per case are shown for white and colored males under 55 years of age in table 18.

Specific causes which have a frequency rate for colored males more than twice that for white males include the following: nonindustrial injuries,

TABLE 18.—*Average annual number of cases per 1,000 males under 55 years of age on account of sickness and nonindustrial injuries disabling for 8 consecutive calendar days or longer, average annual number of days of disability per male, and average number of days per case, by color and cause; experience of male members of sick benefit organizations of seven chromate-producing plants, 1946–50, inclusive.*[1]

[The data include experience of six plants during 1946–50, and one plant during 1949–50]

| Cause | Annual number of cases per 1,000 males | | Annual number of days per male[2] | | Average number of days per case[2] | |
|---|---|---|---|---|---|---|
| | White | Colored | White | Colored | White | Colored |
| Sickness and nonindustrial injuries | 86.2 | 156.7 | 3.05 | 5.29 | 35.4 | 33.8 |
| Nonindustrial injuries | 8.1 | 17.3 | .23 | .48 | 28.4 | 27.7 |
| Sickness | 78.1 | 139.4 | 2.82 | 4.81 | 36.1 | 34.5 |
| Respiratory diseases | 34.3 | 79.5 | .93 | 2.44 | 27.0 | 30.8 |
| Tuberculosis of respiratory system | .7 | 5.3 | .07 | .51 | 98.0 | 98.0 |
| Influenza, grippe | 9.5 | 25.5 | .21 | .57 | 22.2 | 22.5 |
| Bronchitis, acute and chronic | 9.5 | 12.0 | .24 | .26 | 25.3 | 21.6 |
| Pneumonia, all forms | 5.7 | 18.7 | .22 | .56 | 38.2 | 29.7 |
| Diseases of pharynx and tonsils | 3.6 | 6.0 | .08 | .12 | 23.0 | 20.6 |
| Other respiratory diseases | 5.3 | 12.0 | .11 | .42 | 20.0 | 34.8 |
| Digestive diseases | 15.9 | 26.2 | .62 | 1.10 | 38.9 | 41.8 |
| Diseases of stomach except cancer | 4.2 | 3.7 | .15 | .24 | 35.3 | 63.6 |
| D[illegible] and enteritis | 1.8 | 3.7 | .04 | .07 | 24.6 | 18.6 |
| A[illegible] | 4.2 | 3.7 | .13 | .13 | 31.1 | 35.6 |
| Hernia | 3.2 | 9.8 | .18 | .50 | 55.9 | 51.4 |
| Other digestive diseases | 2.5 | 5.3 | .12 | .16 | 46.9 | 29.6 |
| Nonrespiratory-nondigestive diseases | 27.2 | 33.7 | 1.25 | 1.27 | 46.1 | 37.7 |
| Infections and parasitic diseases[3] | 1.8 | 3.0 | .06 | .05 | 36.0 | 17.5 |
| Cancer, all sites | 1.8 | 6.0 | .14 | .43 | 80.6 | 71.6 |
| Rheumatism, acute and chronic | 2.8 | 4.5 | .14 | .17 | 49.9 | 39.0 |
| Neuralgia, neuritis, sciatica | 1.1 | .8 | .06 | .01 | 55.7 | 14.0 |
| Other diseases of nervous system | 2.8 | .8 | .16 | .01 | 55.6 | 11.0 |
| [illegible] art | 3.2 | 2.2 | .23 | .17 | 74.2 | 74.7 |
| [illegible] of circulatory system | 2.8 | 1.5 | .07 | .03 | 23.6 | 22.5 |
| Diseases of genitourinary system | 1.8 | 2.2 | .05 | .03 | 26.0 | 13.0 |
| Diseases of skin | 2.5 | 2.2 | .06 | .03 | 24.6 | 13.0 |
| Diseases of organs of movement except diseases of joints | 1.0 | 4.5 | .02 | .08 | 15.7 | 17.5 |
| All other diseases | 5.6 | 6.0 | .26 | .26 | 47.1 | 42.8 |
| Ill-defined and unknown causes | .7 | 0 | .02 | 0 | 26.5 | — |
| Average number of person-years of membership | 2,832 | 1,334 | 2,832 | 1,334 | 2,832 | 1,334 |

[1] Industrial injuries and venereal diseases are not included.
[2] The number of days of disability is the number of calendar days from the date disability began to the date of return to work, or to the 98th day, except in the event the employee died or was pensioned before the 98th day.
[3] Exclusive of influenza and grippe, respiratory tuberculosis, and venereal diseases.

tuberculosis of respiratory system, influenza and grippe, pneumonia, other respiratory diseases, diarrhea and enteritis, hernia, other digestive diseases, cancer, and diseases of organs of movement except diseases of joints. Diseases which occurred more frequently among the white than the colored include diseases of stomach except cancer, appendicitis, neuralgia, neuritis, sciatica, other diseases of nervous system, diseases of heart, other diseases of circulatory system, and diseases of skin.

With regard to color the number of days of disability per male follows much the same pattern as the frequency rate.

Days per case averaged 35.4 for white males and 33.8 for colored. With the exception of influenza and grippe, other respiratory diseases, diseases of the stomach except cancer, appendicitis, diseases of heart, and diseases of organs of movement except joints, the average days per case for the white are equal to or greater than those for the colored.

### Frequency according to broad cause group by color and age

Table 19 shows the effect of age on the frequency of sickness and nonindustrial injuries. For both colors the sickness rate tends to increase with

TABLE 19.—*Average annual number of cases per 1,000 males on account of sickness and nonindustrial injuries disabling for 8 consecutive calendar days or longer, according to broad cause group, by color and age; experience of male members of sick benefit organizations of seven chromate-producing plants, 1946–50, inclusive.*[1]

[The data include experience of six plants during 1946–50, and one plant during 1949–50]

| Age group | Annual number of cases per 1,000 males | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | Total | White | Colored | Total | White | Colored | Total | White | Colored |
| | Sickness and nonindustrial injuries | | | Nonindustrial injuries | | | Total sickness | | |
| All ages.. | 128.3 | 116.3 | 158.4 | 10.6 | 8.5 | 15.8 | 117.7 | 107.8 | 142.6 |
| Under 35 years.. | 99.5 | 78.9 | 142.9 | 12.7 | 9.9 | 18.7 | 86.8 | 69.0 | 124.2 |
| 35–44 years..... | 89.2 | 71.9 | 129.0 | 10.3 | 6.9 | 18.1 | 78.9 | 65.0 | 110.9 |
| 45–54 years..... | 143.6 | 113.3 | 202.9 | 9.9 | 7.5 | 14.7 | 133.7 | 105.8 | 188.2 |
| 55 years and over | 213.6 | 219.0 | 177.4 | 8.4 | 9.6 | 0 | 205.2 | 209.4 | 177.4 |
| | Respiratory diseases | | | Digestive diseases | | | Nonrespiratory-nondigestive diseases [2] | | |
| All ages.. | 52.5 | 41.5 | 80.2 | 20.9 | 18.8 | 26.1 | 44.3 | 47.5 | 36.3 |
| Under 35 years.. | 46.1 | 31.5 | 76.6 | 21.4 | 16.8 | 31.0 | 19.3 | 20.7 | 16.6 |
| 35–44 years..... | 40.5 | 31.5 | 61.1 | 14.4 | 12.8 | 18.1 | 24.0 | 20.7 | 31.7 |
| 45–54 years..... | 61.9 | 41.1 | 102.7 | 22.3 | 18.7 | 29.3 | 49.5 | 46.0 | 56.2 |
| 55 years and over | 69.1 | 66.2 | 88.7 | 28.3 | 28.9 | 24 2 | 107.8 | 114.3 | 64.5 |
| | Average number of person-years of membership[3] | | | | | | | | |
| All ages.. | 5,121 | 3,663 | 1,458 | 5,121 | 3,663 | 1,458 | 5,121 | 3,663 | 1,458 |
| Under 35 years.. | 1,497 | 1,014 | 483 | 1,497 | 1,014 | 483 | 1,497 | 1,014 | 483 |
| 35–44 years..... | 1,457 | 1,015 | 442 | 1,457 | 1,015 | 442 | 1,457 | 1,015 | 442 |
| 45–54 years..... | 1,212 | 803 | 409 | 1,212 | 803 | 409 | 1,212 | 803 | 409 |
| 55 years and over | 955 | 831 | 124 | 955 | 831 | 124 | 955 | 831 | 124 |

[1] Industrial injuries and venereal diseases are not included.
[2] Ill-defined and unknown causes are included.
[3] Of the 831 white person-years of membership 136 or 16 percent were 65 years and over; of the 124 colored person-years of membership 9 or 7 percent were 65 years and over.

advancing age. Among white males 55 years and over, the nonrespiratory-nondigestive disease rate is more than five times that for persons under 35 years. Among colored males the older group has a frequency rate almost four times that of the younger group. Respiratory diseases are much more common among colored males at each age group, but the relative excess was less among persons 55 years and over.

### Frequency in chromate plants compared with various industries

The sickness experience during 1946–50 for white male chromate workers is compared with a large group of industrial workers (predominantly white) in table 20. The annual number of cases per 1,000 males for sickness was 107.8 for chromate workers and 96.3 for other industrial workers. In

TABLE 20.—*Average annual number of cases per 1,000 males on account of sickness and nonindustrial injuries disabling for 8 consecutive calendar days or longer, by cause; experience of white male members of sick benefit organizations of seven chromate-producing plants in comparison with the experience of male employees in various industries, 1946–50, inclusive.*[1]

[The data include experience of six plants during 1946–50, and one plant during 1949–50]

| Cause | Annual number of cases per 1,000 males | | Number of cases | |
|---|---|---|---|---|
| | Chromate | Various industries [2] | Chromate | Various industries [2] |
| Sickness and nonindustrial injuries | 116.3 | 108.4 | 426 | 112,803 |
| Nonindustrial injuries | 8.5 | 12.1 | 31 | 12,566 |
| Sickness | 107.8 | 96.3 | 395 | 100,237 |
| Respiratory diseases | 41.5 | 34.0 | 152 | 35,406 |
| Tuberculosis of respiratory system | .8 | .6 | 3 | 644 |
| Influenza, grippe | 13.1 | 11.9 | 48 | 12,389 |
| Bronchitis, acute and chronic | 11.7 | 5.5 | 43 | 5,731 |
| Pneumonia, all forms | 6.3 | 4.2 | 23 | 4,407 |
| Diseases of pharynx and tonsils | 3.6 | 3.7 | 13 | 3,865 |
| Other respiratory diseases | 6.0 | 8.1 | 22 | 8,370 |
| Digestive diseases | 18.8 | 17.6 | 69 | 18,356 |
| Diseases of stomach except cancer | 4.9 | 5.5 | 18 | 5,749 |
| Diarrhea and enteritis | 1.9 | 2.3 | 7 | 2,347 |
| Appendicitis | 3.8 | 3.6 | 14 | 3,774 |
| Hernia | 4.4 | 2.7 | 16 | 2,842 |
| Other digestive diseases | 3.8 | 3.5 | 14 | 3,644 |
| Nonrespiratory-nondigestive diseases | 46.7 | 41.6 | 171 | 43,287 |
| Infectious and parasitic diseases [3] | 1.4 | 2.6 | 5 | 2,726 |
| Cancer, all sites | 7.1 | .7 | 26 | 750 |
| Rheumatism, acute and chronic | 4.1 | 4.0 | 15 | 4,186 |
| Neuralgia, neuritis, sciatica | 1.6 | 2.4 | 6 | 2,490 |
| Other diseases of nervous system | 3.5 | 3.7 | 13 | 3,851 |
| Diseases of heart | 7.9 | 4.6 | 29 | 4,739 |
| Other diseases of circulatory system | 6.0 | 6.3 | 22 | 6,591 |
| Nephritis, acute and chronic | .6 | .4 | 2 | 405 |
| Other diseases of genitourinary system | 3.0 | 3.4 | 11 | 3,518 |
| Diseases of skin | 3.0 | 3.6 | 11 | 3,686 |
| Diseases of organs of movement except diseases of joints | 1.9 | 3.3 | 7 | 3,459 |
| All other diseases | 6.6 | 6.6 | 24 | 6,886 |
| Ill-defined and unknown causes | .8 | 3.1 | 3 | 3,188 |
| Average number of person-years of membership | 3,663 | 1,040,707 | 3,663 | 1,040,707 |

[1] Industrial injuries and venereal diseases are not included.
[2] Based on data periodically received by Division of Occuaptional Health, PHS.
[3] Exclusive of influenza and grippe, respiratory tuberculosis, and venereal diseases.

general, the rates for specific causes among chromate workers are not greatly different from those found for other industrial workers. However, it will be observed that illness from cancer appears excessive among chromate workers being 7.1 compared with 0.7 for others. Respiratory diseases are slightly higher among chromate workers, the major part of the excess being due to higher rates for influenza and grippe, bronchitis, acute and chronic, and pneumonia. When cancer is omitted the rate for nonrespiratory-nondigestive diseases is very similar for the two groups, except for a slightly higher heart disease rate among chromate workers.

## *Disability and Deaths from Cancer, 1940-50*

Disability and deaths from cancer, 1940–50, are based on the experience of members of 2 plants during 1940–50, 1 plant during 1943–50, 3 plants during 1946–50, and 1 plant during 1949–50, which yielded a total of 7,818 person-years of membership, of which 5,502 were for white males and 2,316 were for colored males.

### Deaths

During the 11-year period there were 44 deaths from cancer occurring within one year after the chromate worker had ceased employment because of disability due to cancer. An additional four deaths occurred among persons disabled for more than a year. The following table shows for the former group the length of terminal sickness according to age and color.

| Number of days disabled before death | Age at death in years | | | |
|---|---|---|---|---|
| | Total | Under 50 | 50–59 | 60 and over |
| | White males | | | |
| Total | 24 | 9 | 8 | 7 |
| Less than 100 | 12 | 6 | 4 | 2 |
| 100–199 | 10 | 3 | 3 | 4 |
| 200–365 | 2 | 0 | 1 | 1 |
| | Colored males | | | |
| Total | 20 | 10 | 10 | 0 |
| Less than 100 | 10 | 5 | 5 | 0 |
| 100–199 | 7 | 4 | 3 | 0 |
| 200–365 | 3 | 1 | 2 | 0 |

It would appear that half of the white males and half of the colored males died in less than 100 days after becoming disabled. With increasing age there was a tendency for the length of the terminal sickness to increase.

According to site (as recorded on death certificate), respiratory cancers accounted for 16 of the 22 cases lasting less than 100 days, 12 of the 17 cases lasting 100–199 days, and 4 of the 5 cases lasting 200 days and over. Seven persons after being disabled from respiratory cancer lived from 164 to 327 days.

### No record of death during period

There were nine cases of sickness due to cancer originating during 1940–1950 for which there was no record of fatal termination in this period. One case of stomach cancer was reported in 1947 causing an absence of 39 days. In 1949 there was a case of lip cancer causing an absence of 55 days. A gastrointestinal and a respiratory cancer each caused disability lasting beyond the maximum benefit period of 98 days. The worker with the gastrointestinal cancer had not returned to work by 1951, but there was no record of his death. The worker with the respiratory cancer died in 1951. There were 5 cases of disability due to cancer which began during 1950. One man with gastrointestinal cancer returned to work after an absence of 57 days. Another man with cancer of the lip was disabled 11 days. Two men with respiratory cancers and one man with cancer of the prostate were still sick at the end of 1950. Sickness of these men had already lasted 6, 11, and 7 months, respectively.

## *Deaths from Cancer and Other Causes, 1940-50*

Deaths from cancer and other causes, 1940–50, are based on the same plants and the same years as indicated in the previous section; namely, 7,818 person-years of membership. For the period covered by the sickness survey age-specific death rates have been calculated. All rates are expressed as deaths per 100,000 males on an annual basis.

### Death rates per 100,000 males from cancer, by color and age

It will be noted from table 21 that the cancer death rate for all males (562.8) represents 39 percent of the entire rate for all causes (1,458.2). The cancer rate for white males is 32 percent and for colored males is 51 percent of the corresponding total death rates. At ages 45–54 and 55–64 years the cancer death rate among colored males is higher than the rate for all other causes. In the general population of the United States the death rate from cancer had not reached one-fifth of the total rate even in those age groups where cancer is most important.

A racial comparison of cancer death rates shows a white rate of 133.8 for under 35 years of age and 1,579.0 for 65 years of age and over, with no colored deaths observed in these age groups. In the most important productive years, namely, 35–44 and 45–54, the colored cancer rate was more than three times the white rate, and at 55–64 years it is more than double the white rate.

### Death rates per 100,000 males by color and cause

Table 22 gives the number of deaths and death rates per 100,000 male chromate workers by color and specific cause. Of interest is the difference in the classification of causes of death by system for the white and colored males.

Of the 75 deaths among white males 34, or 45 percent, were due to dis-

TABLE 21.—*Average annual number of deaths per 100,000 males from all causes and from cancer, by color and age; experience of male members of sick benefit organizations in seven chromate-producing plants, 1940-50 inclusive.*[1]

[The data include experience of two plants during 1940–50, one plant during 1943–50, three plants during 1946–50, and one plant during 1949–50.]

| Age group | Annual number of deaths per 100,000 males[2] | | | Number of deaths [2] | | | Average number of person-years of membership |
|---|---|---|---|---|---|---|---|
| | All causes | Cancer, all sites | All other causes | All causes | Cancer, all sites | All other causes | |
| | All males | | | | | | |
| All ages | 1,458.2 | 562.8 | 895.4 | 114 | 44 | 70 | 7,818 |
| Under 35 years | 402.7 | 89.5 | 313.2 | 9 | 2 | 7 | 2,235 |
| 35–44 years | 709.2 | 221.6 | 487.6 | 16 | 5 | 11 | 2,256 |
| 45–54 years | 1,908.2 | 928.3 | 979.9 | 37 | 18 | 19 | 1,939 |
| 55–64 years | 3,465.8 | 1,352.5 | 2,113.3 | 41 | 16 | 25 | 1,183 |
| 65 years and over | 5,365.8 | 1,463.4 | 3,902.4 | 11 | 3 | 8 | 205 |
| | White males | | | | | | |
| All ages | 1,363.1 | 436.2 | 926.9 | 75 | 24 | 51 | 5,502 |
| Under 35 years | 334.4 | 133.8 | 200.6 | 5 | 2 | 3 | 1,495 |
| 35–44 years | 517.5 | 129.4 | 388.1 | 8 | 2 | 6 | 1,546 |
| 45–54 years | 1,335.4 | 471.3 | 864.1 | 17 | 6 | 11 | 1,273 |
| 55–64 years | 3,406.8 | 1,102.2 | 2,304.6 | 34 | 11 | 23 | 998 |
| 65 years and over | 5,789.5 | 1,579.0 | 4,210.5 | 11 | 3 | 8 | 190 |
| | Colored males | | | | | | |
| All ages | 1,683.9 | 863.5 | 820.4 | 39 | 20 | 19 | 2,316 |
| Under 35 years | 540.5 | 0 | 540.5 | 4 | 0 | 4 | 740 |
| 35–44 years | 1,126.8 | 422.5 | 704.3 | 8 | 3 | 5 | 710 |
| 45–54 years | 3,003.0 | 1,801.8 | 1,201.2 | 20 | 12 | 8 | 666 |
| 55–64 years | 3,783.8 | 2,702.7 | 1,081.1 | 7 | 5 | 2 | 185 |
| 65 years and over | 0 | 0 | 0 | 0 | 0 | 0 | 15 |

[1] Industrial injuries are not included.
[2] Eleven members who had been disabled more than 1 year and died during 1940–50 are not included. Of these 4 white members died of cancer, 4 white and 3 colored died of other causes.

eases of heart, arteriosclerosis, cerebral hemorrhage and thrombosis, and yielded a total rate of 617.9. Nineteen, or 25 percent, were due to diseases of the respiratory system, including cancer of respiratory system 14, tuberculosis of respiratory system 2, and other respiratory diseases 3. These yielded a total rate of 345.3. Deaths due to diseases of digestive system, including cancer of digestive organs and peritoneum 6, cirrhosis and other diseases of liver 3, and other digestive diseases 2, caused 11 deaths or a rate of 200.0 deaths per 100,000 white males. Of the remaining 11 deaths, 1 was due to cancer of nasopharynx, 1 to cancer of genitourinary organ, 1 to leukemia, 1 to Hodgkin's disease, 1 to nephritis, 1 to alcoholic psychosis, 1 to diabetes, 3 to nonindustrial injuries, and 1 to ill-defined cause.

Of the 39 deaths among the colored 27, or 69 percent, were due to diseases of the respiratory system, including cancer of the respiratory system 18, tuberculosis of the respiratory system 6, and other respiratory diseases 3. The total rate for diseases of the respiratory system, cancerous and noncancerous, amounted to 1,165.8 deaths per 100,000 colored males. Six

TABLE 22.—*Average annual number of deaths per 100,000 males, by color and cause; experience of male members of sick benefit organizations in seven chromate-producing plants, 1940–50, inclusive.*[1]

[The data include experience of two plants during 1940–50, one plant during 1943–50, three plants during 1946–50, and one plant during 1949–50.]

| Cause | Annual number of deaths per 100,000 males [2] | | | Number of deaths [2] | | |
|---|---|---|---|---|---|---|
| | All males | White | Colored | All males | White | Colored |
| All causes | 1,458.2 | 1,363.1 | 1,683.9 | 114 | 75 | 39 |
| Cancer, all sites | 562.8 | 436.2 | 863.5 | 44 | 24 | 20 |
| Respiratory system | 409.3 | 254.4 | 777.2 | 32 | 14 | 18 |
| Digestive organs and peritoneum | 89.5 | 109.1 | 43.1 | 7 | 6 | 1 |
| Buccal cavity and pharynx | 25.6 | 18.2 | 43.2 | 2 | 1 | 1 |
| Genitourinary organs | 12.8 | 18.2 | 0 | 1 | 1 | 0 |
| Leukemia and Hodgkin's disease | 25.6 | 36.3 | 0 | 2 | 2 | 0 |
| Diseases of heart and arteriosclerosis | 383.7 | 454.3 | 215.9 | 30 | 25 | 5 |
| Cerebral hemorrhage and thrombosis | 127.9 | 163.6 | 43.2 | 10 | 9 | 1 |
| Tuberculosis of respiratory system | 102.3 | 36.3 | 259.1 | 8 | 2 | 6 |
| Other respiratory diseases | 76.8 | 54.6 | 129.5 | 6 | 3 | 3 |
| Cirrhosis and other diseases of liver | 38.3 | 54.5 | 0 | 3 | 3 | 0 |
| Other digestive diseases | 25.6 | 36.4 | 0 | 2 | 2 | 0 |
| All other diseases | 64.0 | 72.7 | 43.2 | 5 | 4 | 1 |
| Nonindustrial injuries | 76.8 | 54.5 | 129.5 | 6 | 3 | 3 |

1 Industrial injuries are not included.

2 Eleven members who had been disabled more than 1 year and died during 1940–50 are not included. Of these, 4 white members died of cancer, 2 white and 2 colored died of tuberculosis of respiratory system, 2 white died of heart disease, and 1 colored died of pneumonia.

NOTE.—Average number of person-years of membership; white, 5,502, colored, 2,316.

deaths due to diseases of heart, arteriosclerosis, cerebral hemorrhage and thrombosis, yielded a total rate of 259.1 per 100,000 colored males. Of the remaining 6 deaths 1 was due to cancer of the digestive system, 1 to cancer of nasopharynx, 1 to meningitis, and 3 to nonindustrial injuries.

## *Comparison of Mortality Experience for Chromate Workers with Total Males in United States, 1940-48*

It is apparent from the foregoing discussion that respiratory cancer occurred above normal frequency among chromate workers. Hence, an estimate of the excess in the number of deaths should be of interest. In table 23, death rates based upon the white and colored male population of the United States, 1940–48, are compared with death rates for similar age groups among chromate workers.

During the 9-year period, 1940–48, two plants reported during 1940–48; one plant, during 1943–48; and three plants, during 1946–48. The number of person-years of membership for each of the 5 age groups is shown in the table on page 55.

Only in the oldest age group, 65–74 years, in the United States population were there an appreciable number of males not in the labor force; thus the death rates for males 15–74 are based essentially on the working population. Because of the small size of the chromate person-years of membership,

TABLE 23.—*Number of deaths from cancer and all causes except cancer, by color and age, among male members of sick benefit organizations in six chromate-producing plants compared with the expected number based on the average death rate for the United States, 1940–48, inclusive.*[1]

[The chromate data include experiences of two plants during 1940–48, one plant during 1943–48, three plants during 1946–48.]

| Cause and age group | Ratio of actual to expected number (Chromate) | Number of deaths: Actual (Chromate) | Number of deaths: Expected [2] (Chromate) | Annual number of deaths per 100,000 males: Chromate | Annual number of deaths per 100,000 males: U.S. [3] |
|---|---|---|---|---|---|
| **All males** | | | | | |
| *All causes, except cancer* | | | | | |
| Total, 15–74 years | 1.16 | 51 | 44.0 | 923.6 | 797.5 |
| 15–34 years | 2.63 | 5 | 1.9 | 317.6 | 121.8 |
| 35–44 years | 1.69 | 10 | 5.9 | 618.8 | 366.6 |
| 45–54 years | 1.17 | 15 | 12.8 | 1,063.1 | 905.5 |
| 55–64 years | 1.10 | 17 | 15.4 | 2,135.7 | 1,940.7 |
| 65–74 years | .75 | 4 | 5.3 | 3,200.0 | 4,238.7 |
| *Cancer, all sites* | | | | | |
| Total, 15–74 years | 4.44 | 32 | 7.2 | 579.5 | 130.3 |
| 15–34 years | 20.00 | 2 | .1 | 127.1 | 9.1 |
| 35–44 years | 5.00 | 3 | .6 | 185.6 | 39.3 |
| 45–54 years | 7.50 | 15 | 2.0 | 1,063.1 | 140.4 |
| 55–64 years | 3.45 | 10 | 2.9 | 1,256.3 | 363.6 |
| 65–74 years | 2.22 | 2 | .9 | 1,600.0 | 757.9 |
| *Cancer of respiratory system, except larynx* | | | | | |
| Total, 15–74 years | 28.89 | 26 | .9 | 470.8 | 16.7 |
| 15–44 years | 40.00 | 4 | .1 | 125.4 | 2.5 |
| 45–54 years | 30.00 | 12 | .4 | 850.5 | 25.8 |
| 55–74 years | 20.00 | 10 | .5 | 1,085.8 | 57.2 |
| *Cancer, all other sites* | | | | | |
| Total, 15–74 years | .95 | 6 | 6.3 | 108.7 | 113.6 |
| 15–44 years | 2.00 | 1 | .5 | 31.3 | 15.7 |
| 45–54 years | 1.88 | 3 | 1.6 | 212.6 | 114.6 |
| 55–74 years | .49 | 2 | 4.1 | 217.1 | 448.5 |
| **White males** | | | | | |
| *All causes, except cancer* | | | | | |
| Total, 15–74 years | 1.24 | 36 | 29.1 | 941.6 | 760.6 |
| 15–34 years | 2.00 | 2 | 1.0 | 192.1 | 98.8 |
| 35–44 years | 1.76 | 6 | 3.4 | 550.0 | 312.0 |
| 45–54 years | 1.07 | 8 | 7.5 | 870.5 | 816.5 |
| 55–64 years | 1.31 | 16 | 12.2 | 2,427.9 | 1,857.3 |
| 65–74 years | .85 | 4 | 4.7 | 3,539.8 | 4,167.1 |
| *Cancer, all sites* | | | | | |
| Total, 15–74 years | 2.94 | 15 | 5.1 | 392.4 | 133.6 |
| 15–34 years | 20.00 | 2 | .1 | 192.1 | 9.2 |
| 35–44 years | 2.50 | 1 | .4 | 91.6 | 38.3 |
| 45–54 years | 3.08 | 4 | 1.3 | 435.3 | 137.1 |
| 55–64 years | 2.50 | 6 | 2.4 | 910.5 | 365.7 |
| 65–74 years | 2.22 | 2 | .9 | 1,769.9 | 774.8 |
| *Cancer of respiratory system, except larynx* | | | | | |
| Total, 15–74 years | 14.29 | 10 | .7 | 261.6 | 17.4 |
| *Cancer, all other sites* | | | | | |
| Total, 15–74 years | 1.14 | 5 | 4.4 | 130.8 | 116.2 |
| **Colored males** | | | | | |
| *All causes, except cancer* | | | | | |
| Total, 15–74 years | .77 | 15 | 19.5 | 882.9 | 1,149.7 |
| 15–34 years | 1.76 | 3 | 1.7 | 562.9 | 316.2 |
| 35–44 years | .87 | 4 | 4.6 | 761.9 | 869.7 |
| 45–54 years | .77 | 7 | 9.1 | 1,422.7 | 1,845.6 |
| 55–64 years | .24 | 1 | 4.1 | 929.9 | 2,974.0 |
| 65–74 years | ...... | 0 | .6 | 0 | 5,206.5 |
| *Cancer, all sites* | | | | | |
| Total, 15–74 years | 10.00 | 17 | 1.7 | 1,000.6 | 98.5 |
| 15–34 years | ...... | 0 | ..... | 0 | 8.6 |
| 35–44 years | 6.67 | 2 | .3 | 381.0 | 49.3 |
| 45–54 years | 12.22 | 11 | .9 | 2,235.8 | 174.4 |
| 55–64 years | 8.00 | 4 | .5 | 2,919.7 | 336.6 |
| 65–74 years | .... | 0 | .1 | 0 | 529.5 |
| *Cancer of respiratory system, except larynx* | | | | | |
| Total, 15–74 years | 80.00 | 16 | .2 | 941.7 | 10.0 |
| *Cancer, all other sites* | | | | | |
| Total, 15–74 years | .67 | 1 | 1.5 | 58.9 | 88.5 |

[1] Violent and accidental deaths are not included.

[2] The average death rate for the United States for the 9 years, 1940–48, multiplied by the appropriate person-years of membership in the chromate plants.

[3] Data are derived from references 35A, 35B and 35C.

attention should be centered on general trends rather than particular age specific rates.

| Color | Total 15–74 | Age group (years) | | | | |
|---|---|---|---|---|---|---|
| | | 15–34 | 35–44 | 45–54 | 55–64 | 65–74 |
| | Average number of person-years of membership | | | | | |
| All males | 5,522 | 1,574 | 1,616 | 1,411 | 796 | 125 |
| White males | 3,823 | 1,041 | 1,091 | 919 | 659 | 113 |
| Colored males | 1,699 | 533 | 525 | 492 | 137 | 12 |

## Deaths among all males

In table 23 it will be noted that among all males the ratio of actual to expected number of deaths from all causes except cancer declines steadily with advancing age until it becomes favorable for chromate workers. For cancer, all sites, the actual number of deaths was approximately four and one-half times the number that would have been expected, based upon occurrence in the total U. S. male population. When cancer of the respiratory system was observed separately, the excess for chromate workers was greatly increased. Nearly 29 times as many deaths due to respiratory cancer were found as would have been expected. For three ascending age groups, respiratory cancer was 40, 30, and 20 times as common among male chromate workers as among other males. All other types of cancer failed to show an excess among chromate workers.

## Deaths among white and colored males

A racial comparison of respiratory cancer deaths shows that the ratio of actual to expected number was 14.29 for whites and 80.00 for colored. On the other hand, cancer of all other sites was about what would be expected for whites (1.14), but was less frequent than expected for colored chromate workers (0.67).

For all causes of death except cancer the white death rate (941.6) was somewhat over, and the colored death rate (882.9) was somewhat under, that found in the general population (760.6 and 1,149.7, respectively). With advancing age, chromate workers of both colors showed a decreasing trend of actual to expected deaths from causes other than cancer.

The number of recorded deaths from respiratory cancer among the chromate workers is minimal for the following reasons:

1. Deaths of employees who were not members of a sick benefit association are not included.
2. Persons who worked in chromate but left the industry prior to their terminal illness are not included.
3. Members who died over a year after onset of disability due to cancer are not included.
4. Several members who had a clinical course consistent with the presence of cancer of the respiratory tract are not included because cancer was not recorded on their death certificates.
5. Some members whose deaths were not recorded as cancer died without a complete medical examination or biopsy.

## *Summary*


The morbidity and mortality experience of male members of sick benefit associations in 7 chromate-producing plants is described. Sickness among a group of white male chromate workers was compared with that of a large group of industrial workers. It was observed that the annual number of cases of sickness and nonindustrial injuries per 1,000 white males was 116.3 for chromate workers and 108.4 for other industrial workers.

For all specific causes of morbidity except cancer the chromate workers had frequency rates that were not greatly different from other workers. Cancer, with a rate of 7.1 compared with 0.7, stands out as markedly in excess for chromate workers.

The frequency of sickness among chromate workers has shown a trend downward during the past 5 years. The most marked decline has occurred among respiratory diseases.

Colored chromate workers under 55 years of age had a higher rate for sickness and nonindustrial injuries than had the white workers. For cases lasting 8 days or longer the former had a frequency rate of 156.7 per 1,000 compared with 86.2 for the latter.

During the 9-year period, 1940–48, the average annual death rate per 100,000 males aged 15–74 years for all causes was 1,503.1 for chromate workers and 927.8 for the corresponding male population of the United States; for all causes except cancer the rates were 923.6 and 797.5, respectively.

For cancer, all sites, the actual number of deaths of chromate workers was approximately four and one-half times the number that would have been expected had the cancer rates for all males in the United States prevailed. When cancer of the respiratory system was observed separately for chromate workers, nearly 29 times as many deaths as were expected were found. All other types of cancer failed to show an excess among chromate workers. A racial comparison of respiratory cancer deaths showed that the ratio of actual to expected number was 14 for whites and 80 for colored; for cancer, all other sites, the two ratios were markedly lower.

# MEDICAL SURVEY

The medical studies were undertaken to determine the present health status of the chromate workers and to evaluate the effects of the working environment on their health. With this objective in mind, the scope of the examination was made broad enough so as to consider other factors which might influence the workers' health. Thus, histories of past and present occupations, familial diseases, past illnesses, and the workers' personal habits were included in the examination.

The plant medical program, including the preplacement and periodic examinations, was evaluated. These examinations might influence the composition of the group surveyed. For example, where there were periodic X-ray examinations of the chest we would expect to find a lower prevalence of pulmonary tuberculosis and other pulmonary findings since persons so affected would be screened out.

The medical examination reviewed the patient as a whole, giving especial attention to certain organ systems which it appeared would be most likely to be involved in exposure to chromates. Radiologists and otorhinolaryngologists participated in certain phases of the survey. For purposes of comparison two industrial plants were selected which had similar working populations and somewhat similar processes, but with no exposure to chromates.

## *Personnel and Facilities*

The medical field unit consisted of a physician in charge and the examining team. The physician in charge was responsible for all activities in the field during the course of the survey. This consisted of supervising the examining team, arranging entries into plants scheduled for study, keeping the field unit supplied with materials, and arranging for any special studies which seemed to be indicated as the survey progressed.

The examining team was composed of a physician, dentist, nurse and a laboratory technician. The physician obtained the medical history and carried out the physical examination. The dentist's task was to examine the teeth and oral structures.

The nurse took the occupational and personal history, and performed other procedures. In three of the plants a second nurse was made available to the team from a state health agency. She assisted the regular nurse in taking all but the occupational history. The same laboratory technician was used for the entire study. Therefore, it is to be noted that changes in

personnel doing different portions of the examination were kept to a minimum.

The examinations were carried out on the plant premises in every instance. The nature of the examinations required facilities such as an adequate supply of running water, a number of electrical outlets and sufficient space. Management was very cooperative in arranging suitable accommodations.

The workers and their unions were notified that the examinations were to be done on a voluntary basis. Plant foremen, personnel managers, or dispensary chiefs cooperated by arranging time schedules for the men to come in for examination. Men from all shifts were examined. In all plants surveyed over 95 percent of the workers presented themselves for examination. The time the worker spent being examined varied from 30–50 minutes, with an average of about 45 minutes.

## *Medical Examination*

The nurse recorded the personal and occupational histories, temperature, pulse rate, respiration, height, weight, and lung vital capacity measurements. The last measurements were made with a Collins water-replacement type spirometer.

The laboratory technician took samples and made tests of the blood and urine. The urine was studied for: (1) Specific gravity; (2) glucose using Benedict's qualitative solution; (3) albumin by sulfosalicylic acid method, comparing with Kingsbury-Clark albuminometric standards; (4) urinary sediment—by examining microscopically the uncentrifuged sediment. On the first 100 men this latter procedure was done only when the urine was positive for albumin; after that it was done routinely. The finger prick method was used to obtain blood for red blood cell counts, white blood cell counts, hemoglobin values, and for blood smears. The counts were done by standard methods. The hemoglobin was determined with a Spencer hemoglobinometer. Wright's stain was used to prepare the slides for differential counting. Two hundred cells were counted.

Ten to 15 cc of blood were drawn from the antecubital vein. About one-third of this was placed in a clean test tube for the serological test for syphilis. The rest was placed in a small bottle containing a dry anticoagulant (6 mg of ammonium oxalate and 4 mg of potassium oxalate). From this bottle blood was taken for determination of the sedimentation rate and hematocrit.

The sedimentation rates were determined by using Wintrobe tubes with readings taken at the end of one hour. These figures were corrected later for the hematocrit readings. The hematocrit determinations were obtained by centrifuging the above Wintrobe tubes for a period of 30 minutes at approximately 2,500 r. p. m.

The blood indices—the mean corpuscular volume, mean corpuscular hemoglobin, and the mean corpuscular hemoglobin concentration—were

obtained from red blood cell count, hemoglobin, and hematocrit by using a suitable nomogram.

Chest X-rays were taken on all workers examined. All exposures were made on 14″ x 17″ films for one-half second at 72″ distance using appropriate voltage and amperage dependent upon the A–P diameter of the chest. In all plants except one, the films were taken on one machine, a portable Picker unit. This plant had its own stationary X-ray machine in the dispensary, and all films were taken on that machine by their technician. With a few exceptions, all films turned out to be of satisfactory quality.

In addition to the foregoing, other procedures of a more specialized nature were carried out on smaller groups of workers. In one of the plants a dermatologist of the Division of Occupational Health studied the skin effects of exposure to chromium compounds. This consisted of examining and describing cutaneous lesions among the chromate workers and patch testing these individuals to determine whether sensitivity existed to chromium.

Urine samples for analysis of chromium were obtained from a representative sample of the men in some of the plants. Special precautions were taken to avoid contamination of the specimens by collecting them at the end of the work period after the men had showered and changed clothes. In addition, at one of the plants urine was collected for analysis for coproporphyrin content.

Blood serum was obtained on 60 workers in one plant for determination of mucoprotein and polysaccharide levels, factors which are often affected in the presence of liver, kidney or neoplastic disease.

## *Characteristics of Examined Workers*

### Birthplace

Birthplace gives some indication of the relative stability of an employed population. Among chromate workers it was found that 33.6 percent were born in the state where they were working at the time of the medical examination. This compares with 31.0 percent among a group of foundry workers. For both industries there was a much higher percentage of native state workers among the whites than among the colored.

Only 5 out of 104 colored workers in New Jersey chromate plants were born within that State. One-half of the out-of-state colored employees came from Georgia or South Carolina. Seventeen percent of the Maryland colored workers were from that State and 88 percent of the out-of-state workers were from North and South Carolina and Virginia.

A third of the white chromate workers were born abroad. Variation among plants was great in this respect. In New York and Maryland 9 percent were born outside the United States, but in New Jersey foreign-born whites accounted for 45 percent. Of these 72 percent were born in Northern Europe, principally Poland.

## Color

Colored males, all Negroes, constituted 37 percent of the total persons examined in chromate plants. Eighty-six percent of all colored males were found in the two largest plants where they represented 46 percent and 37 percent of the workers.

## Age

Table 24 shows the age distribution of the chromate population studied. The median age was 43.1 years which may be compared with a median of 40.7 years for foundry workers *(79)* and 43.0 years for open-hearth steel workers *(80)*. The median age for white and colored chromate workers was 43.9 and 40.5, respectively. Among chromate workers 34.2 percent were 50 years of age or over which is slightly higher than the 32.6 percent for foundry workers.

TABLE 24.—*Age distribution of chromate workers, by color.*

| Age group (years) | Number | | | Percent | | |
|---|---|---|---|---|---|---|
| | Total | White | Colored | Total | White | Colored |
| All ages | 897 | 653 | 244 | 100.0 | 100.0 | 100.0 |
| Under 20 | 1 | 1 | 0 | .1 | .1 | 0 |
| 20–24 | 40 | 34 | 6 | 4.5 | 5.2 | 2.5 |
| 25–29 | 89 | 58 | 31 | 9.9 | 8.9 | 12.7 |
| 30–34 | 121 | 84 | 37 | 13.5 | 12.9 | 15.2 |
| 35–39 | 129 | 84 | 45 | 14.4 | 12.9 | 18.4 |
| 40–44 | 112 | 83 | 29 | 12.5 | 12.7 | 11.9 |
| 45–49 | 98 | 55 | 43 | 10.9 | 8.4 | 17.6 |
| 50–54 | 100 | 70 | 30 | 11.1 | 10.7 | 12.3 |
| 55–59 | 95 | 79 | 16 | 10.6 | 12.1 | 6.6 |
| 60–64 | 72 | 68 | 4 | 8.0 | 10.4 | 1.6 |
| 65 or over | 40 | 37 | 3 | 4.5 | 5.7 | 1.2 |

A somewhat different age pattern was observed for white and colored workers. There was a larger proportion of colored in the age groups from 45 to 55 years, and a much larger proportion of the whites in the group from 55 years of age and over. The percentage under 30 years of age showed slight difference by color.

In regard to older white workers these plants fall into two distinct groups. Plants in Maryland and New York had less than 12 percent 55 years of age and over, while the four plants in New Jersey had a range of 35 to 46 percent in this age group. Both plants with a large colored population had about the same age distribution for colored males.

## Marital status

Eighty-six percent of the workers were married, 8 percent were single, and 6 percent were widowed, separated or divorced. The percent married was slightly greater than among other industrial groups recently studied by the Public Health Service. Among white and colored males 86 and 85 percent, respectively, were married.

## Personal habits

### *Alcohol*

Accurate data on the consumption of alcoholic beverages are difficult to secure. The percentage who denied the use of alcohol, 41 for whites and 61 for colored, is considerably higher than observed among foundrymen. More white and less colored nonchromate workers said they did not use alcohol.

For those who admitted the use of alcohol an attempt was made to make a rough estimate of the number of ounces consumed daily. Among white males 42 percent consumed less than 1 ounce, 39 percent from 1 to 2.9 ounces, and 19 percent 3 ounces or over. The corresponding percentages for colored males were 34, 52, and 14, respectively. Apparently the colored fall less in the extremes of light or heavy drinking.

### *Tobacco*

Smoking was reported by 81.1 percent of the chromate workers and 79.4 percent of the foundrymen. A slightly higher percentage of colored chromate workers smoked than did white workers, 84.0 percent compared with 80.1 percent, respectively. A smaller percentage of both white and colored nonchromate workers smoked.

Tobacco users were separated according to the amount they smoked. Among the white males it was found that 19.9 percent did not smoke, 19.7 percent were light or moderate smokers, 28.4 percent were heavy smokers, and 32.0 percent were very heavy smokers. Corresponding percentages for colored males were 16.0, 42.2, 29.5 and 12.3. It would appear that white males smoked more heavily than colored males.

With advancing age a smaller percentage of both white and colored workers reported smoking. This trend was also observed in the foundry industry.

### *Baths*

Daily baths were reported by 42.9 percent of the chromate workers and by 39.6 percent of the nonchromate workers, which is less favorable than the 53.3 percent reported by foundry workers.

As observed in other recent studies of industrial workers, less frequent baths were taken in the older age groups by white males. After 25 years of age the percentage taking daily baths declined from 54.6 percent at the age 25–34 to 17.0 percent at age 55 years and over. At the same time persons with three or less baths per week increased from 7.1 percent to 30.2 percent.

There were great plant differences in the percent of persons taking daily baths. This practice ranged from 6.1 percent to 78.8 percent. There was a direct relation between bathing facilities provided by the plant and the number of baths taken per week. In two plants three baths or less per week were taken by more than a fourth of the white workers, whereas in a plant which stressed cleanliness, less than 2 percent fell within this group.

### Familial history of disease

A familial history (parents, brothers and sisters) of allergy was reported by 11.8 percent of the white males and 7.4 percent of the colored males. Cancer was reported in 9.9 percent of the white families and 2.9 percent of the colored families. Two percent of the white workers had records of both cancer and allergy in their families. Differences according to plant were not great. In regard to a history of both allergy and cancer white and colored nonchromate workers showed little difference from chromate workers.

### Occupational history

Among chromate workers there was a relatively small percentage with no previous experience other than agriculture, forestry or fishing. This class represented 4.1 percent of the chromate workers compared with 23.1 percent of the foundrymen. Trade or service occupations had previously been followed by 14.4 percent of the chromate workers and 19.4 percent of the foundrymen. A much larger percentage of the chromate workers had previous experience in metal working industries than had foundrymen, 27.7 percent compared with 14.0 percent. Experience in some other chemical industry was reported by 8.2 percent of the chromate workers. Exposure in dusty trades was found among 4.2 percent. The remaining workers had experience in a variety of industries not known to involve harmful exposures.

White and colored workers had little difference in experience with regard to trade or service and dusty trades. The proportion of white persons with metal working and chemical experience was much greater, while colored persons appeared more frequently in the "all other" classification.

### Years in chromate work

A considerable number of workers have been employed in the chromate industry for a long period. Table 25 shows that 5.8 percent have been employed 30 years or more, 18.7 percent 20 years or more, and 41.2 percent 10 years or more. White males have worked in chromate longer than colored males. For example, 21.8 percent of the former but only 10.2

TABLE 25.—*Distribution of chromate workers, according to years in chromate industry, by color.*

| Years in chromate industry | Number | | | Percent | | |
|---|---|---|---|---|---|---|
| | Total | White | Colored | Total | White | Colored |
| Total | 897 | 653 | 244 | 100.0 | 100.0 | 100.0 |
| Less than 5 | 346 | 244 | 102 | 38.6 | 37.5 | 41.8 |
| 5–9 | 182 | 112 | 70 | 20.3 | 17.2 | 28.7 |
| 10–14 | 101 | 72 | 29 | 11.2 | 10.9 | 11.9 |
| 15–19 | 101 | 83 | 18 | 11.2 | 12.6 | 7.4 |
| 20–24 | 65 | 52 | 13 | 7.3 | 8.0 | 5.3 |
| 25–29 | 50 | 43 | 7 | 5.6 | 6.6 | 2.9 |
| 30–34 | 40 | 35 | 5 | 4.5 | 5.4 | 2.0 |
| 35–39 | 7 | 7 | 0 | .8 | 1.1 | ........ |
| 40–44 | 5 | 5 | 0 | .5 | .7 | ........ |

percent of the latter, have worked in chromate 20 years or more.

Considering white males only there appear to be great plant differences in length of employment. In two plants the 20 year or longer group represented less than 7 percent, while in one plant half of the workers had stayed that long.

## Present occupation

Operations in the various chromate plants were not identical, but certain broad occupational groupings are possible. Table 26 shows the age distribution of white and colored workers in seven operations. The largest percentage of white males under 35 years of age were found in special processes. Younger colored persons were most common in the liquor room. The occupations where the largest percentage of workers 55 years of age or over were found are kiln room for white and primary leach and residue drying for colored workers.

TABLE 26.—*Chromate workers according to present occupational groups, by age and color.*

| Occupational group | White | | | | Colored | | | |
|---|---|---|---|---|---|---|---|---|
| | Total | Age (years) Under 35 | 35–54 | 55 or over | Total | Age (years) Under 35 | 35–54 | 55 or over |
| | Number | | | | | | | |
| Total | 653 | 177 | 292 | 184 | 244 | 74 | 147 | 23 |
| Mill room | 47 | 6 | 20 | 21 | 23 | 4 | 16 | 3 |
| Kiln room | 37 | 5 | 14 | 18 | 27 | 8 | 16 | 3 |
| Primary leach and residue drying | 21 | 4 | 8 | 9 | 51 | 12 | 32 | 7 |
| Liquor room | 116 | 39 | 56 | 21 | 48 | 21 | 22 | 5 |
| Special processes | 69 | 25 | 27 | 17 | 33 | 11 | 22 | 0 |
| Maintenance | 181 | 60 | 86 | 35 | 13 | 3 | 10 | 0 |
| Other | 182 | 38 | 81 | 63 | 49 | 15 | 29 | 5 |
| | Percent | | | | | | | |
| Total | 100 | 27.1 | 44.7 | 28.2 | 100 | 30.3 | 60.2 | 9.5 |
| Mill room | 100 | 12.7 | 42.6 | 44.7 | 100 | 17.4 | 69.6 | 13.0 |
| Kiln room | 100 | 13.5 | 37.8 | 48.7 | 100 | 29.6 | 59.3 | 11.1 |
| Primary leach and residue drying | 100 | 19.0 | 38.1 | 42.9 | 100 | 23.5 | 62.8 | 13.7 |
| Liquor room | 100 | 33.6 | 48.3 | 18.1 | 100 | 43.8 | 45.8 | 10.4 |
| Special processes | 100 | 36.3 | 39.1 | 24.6 | 100 | 33.3 | 66.7 | 0 |
| Maintenance | 100 | 33.2 | 47.5 | 19.3 | 100 | 23.1 | 76.9 | 0 |
| Other | 100 | 20.9 | 44.5 | 34.6 | 100 | 30.6 | 59.2 | 10.2 |

Occupations in the mill room are concerned with crushing and drying the raw chromite ore and with the addition of lime and soda ash. In this area were found 7.2 percent of the white and 9.4 percent of the colored workers. The kiln room where the ore is roasted in rotary kilns is one of the hottest areas in a chromate plant. A relatively small number worked near the kilns, namely 5.7 percent of the whites and 11.1 percent of the colored. In primary leach and residue drying there were 3.2 percent of the white workers and 20.9 percent of the colored workers. By the time the material reached the liquor area all processes were dealing with fluids. The per-

centages for white and colored workers were 17.8 and 19.7. Special processes carried on only in certain plants included the manufacture of chemically-free chrome compounds, aluminum hydrate, potassium chromate, tanning compounds, and chrome oxide. Here worked 10.5 percent of the white and 13.5 percent of the colored employees. Maintenance was largely performed by white workers, 27.7 percent against 5.3 percent colored. The classification "other" included primarily office, laboratory and outside yard workers. Only among this group and the maintenance workers did the white males show a higher percentage. Colored males were in excess in each of the other process operations.

## *Medical Findings*

### Previous illnesses

A relatively small proportion of the workers questioned reported any type of respiratory disease since entering the chromate industry. Previous attacks of pneumonia were recalled by 5.8 percent of the white males and 12.7 percent of the colored males. Among nonchromate workers the percentages were 1.4 and 1.2, respectively. Two percent of all chromate males reported pleurisy and 0.8 percent bronchial asthma. Experience of the nonchromate group was not greatly different. Previous attacks of heart disease were mentioned by 1.7 percent of the chromate workers. Chronic bronchitis was reported by 2.5 percent of white chromate workers as compared with 5.0 percent of white ferrous foundry workers *(79)* and by 1.6 percent of the colored chromate workers as compared with 1.3 percent of the colored ferrous foundry workers. Among white nonchromate workers 1.4 percent had had this disease but there had been no case among colored workers in this industrial group.

Almost an equal proportion of the white and colored chromate workers, 27.1 and 27.4 percent, said they had had no colds during the past year. However, 13.0 percent of the white and 21.1 percent of the colored chromate workers had five or more colds during the year. For both colors, the nonchromate workers had a slightly more unfavorable experience with respect to colds.

### Weight deviation

The percent deviation of each chromate worker's weight from the weight of men of similar height and age was calculated from life insurance data *(81)*. Chromate workers were found to have about the same weight distribution as foundry workers and open-hearth steel workers. When chromate workers were compared with the nonchromate workers included in this study, colored males in both groups had nearly the same weight distribution but white male chromate workers were slightly lighter than the white nonchromate workers.

## Head

### *Eyes*

Accidental splashes of chromium compounds into the eyes have been observed in these plants. Such accidents are reported *(12, 13)* to have caused severe chemical burns, increasing in depth and area in the course of time. They often result in chronic deep keratitis, which may last for months. In some cases the iris and ciliary bodies have been damaged. However, this study is principally concerned with the chronic effects which the chromates may have on the eyes, excluding such accidental injuries.

Previous investigations by Hermanni *(17)*, Ranelletti *(20)*, Garcia-Sola *(26)*, have found that the eye was chronically affected by this industry. Naidu and Rao *(8)* reported that in 152 chromate workers 10 percent had eye symptoms.

Table 27 compares eye findings and symptoms for chromate and nonchromate workers, grouped as to color. The most frequent finding was congestion of the conjunctiva which was found in 44.7 percent of white and in 22.5 percent of colored chromate workers compared with 35.1 and 17.3 percent, respectively, of nonchromate workers. Discharge was found among less than 5 percent of any group of workers. Corneal scarring was also relatively unimportant, never reaching 4 percent of the workers examined. Grouping all abnormal eye findings together, 46.6 percent of the white chromate workers were affected compared with 25.4 percent of the colored chromate workers. For nonchromate workers the proportion with findings were slightly lower, namely, 36.5 percent and 22.2 percent, respectively.

TABLE 27.—*Selected eye findings and symptoms among chromate and nonchromate workers, by color.*

| Eye findings and symptoms | Number | | | Percent | | |
|---|---|---|---|---|---|---|
| | Total | White | Colored | Total | White | Colored |
| | Chromate | | | | | |
| Total workers | 897 | 653 | 244 | | | |
| *Findings:* | | | | | | |
| Congestion of conjunctiva | 347 | 292 | 55 | 38.7 | 44.7 | 22.5 |
| Discharge | 29 | 29 | 0 | 3.2 | 4.4 | 0 |
| Cornea scar | 21 | 13 | 8 | 2.3 | 2.0 | 3.3 |
| Any abnormal findings | 366 | 304 | 62 | 40.8 | 46.6 | 25.4 |
| *Symptoms:* | | | | | | |
| Epiphora and burning | 152 | 102 | 50 | 17.0 | 15.6 | 20.5 |
| Any symptom | 183 | 125 | 58 | 20.5 | 19.2 | 23.8 |
| | Nonchromate | | | | | |
| Total workers | 155 | 74 | 81 | | | |
| *Findings:* | | | | | | |
| Congestion of conjunctiva | 40 | 26 | 14 | 25.8 | 35.1 | 17.3 |
| Discharge | 2 | 0 | 2 | 1.3 | 0 | 2.5 |
| Cornea scar | 4 | 1 | 3 | 2.6 | 1.4 | 3.7 |
| Any abnormal findings | 45 | 27 | 18 | 29.0 | 36.5 | 22.2 |
| *Symptoms:* | | | | | | |
| Epiphora and burning | 35 | 8 | 27 | 22.6 | 10.8 | 33.3 |
| Any symptom | 37 | 10 | 27 | 23.9 | 13.5 | 33.3 |

Outstanding among the complaints were epiphora and burning. They appear together or singly in 15.6 percent of white and 20.5 percent of colored chromate workers and in 10.8 percent of the white and 33.3 percent of the colored nonchromate workers. The last group had a high rate possibly due to their dusty working environment. The white nonchromate group, not exposed to heavy dust concentration, made a slightly more favorable showing than the white chromate workers. The percentage with any abnormal symptom was 19.2 for white chromate workers and 13.5 for white nonchromate workers.

Congestion of the conjunctiva of white chromate workers does not increase appreciably with age (42.2 percent under 45 years and 47.2 percent 45 years of age or over). In colored chromate workers the corresponding percentages are 21.6 and 22.9. This suggests that irritation to the eye is limited and is not aggravated by length of employment.

### *Nose*

*Prevalence of lesion.*—Numerous reports dating back to the initial report by Cumin *(1)* in 1827 have described the occurrence of nasal mucosal ulceration and perforation in chromate workers.

A summary of some of the previous investigations of workers exposed to chromates who had nasal ulceration or perforation is tabulated as follows:

| Investigator and reference number | Year | Number of men examined | Percent with septal ulceration | Percent with septal perforation |
|---|---|---|---|---|
| Hermanni (Ger.) (17) | 1901 | 257 | 33.5 | 41.6 |
| Legge (Eng.) (4) | 1902 | 176 | 11.3 | 71.6 |
| Fischer (Ger.) (16) | 1902 | 228 | 4.8 | 22.8 |
| Ranelletti (Ital.) (20) | 1919 | 69 | 55.0 | 8.6 |
| Schwartz (Ger.) (9) | 1922 | 210 | 63.8 | 13.3 |
| Wilensky (USSR) (23) | 1924 | 278 | 17.6 | 33.1 |
| Med. Insp. Fac. (Eng.) (5) | 1930 | 223 | 35.0 | 17.0 |
| Machle and Gregorius (U. S.) (35) | 1948 | 590 | 11.7 | 32.2 |
| Mancuso (U. S.) (34) | 1951 | 97 | 6.2 | 62.9 |

In this present survey of six chromate-producing plants in the United States, a total of 897 workers were examined of whom 509, or 56.7 percent, had perforation of the nasal septum. When the figures of Mancuso *(34)*, which represent the remaining chromate-producing plant, are added there are 994 workers engaged in the production of chromates in the United States, of whom 570 had perforation of the nasal septum. However, with labor turnover and the improvement in methods of control due to change in processes, and better industrial hygiene practices, these figures will be subject to change.

*Pathological considerations.*—Immediately posterior to the nares the dilated portion of the nasal fossa is lined with a squamous type of epithelium which differs slightly from, but is continuous with, the integument covering the nose externally. Beyond this point there is a transition from this modi-

fied skin type of epithelium to the respiratory mucous membrane type which lines the nasal fossa proper. This latter epithelium is characterized in part by cells which have freely-moving hair-like cilia. Interspersed among these cells are other cells and glands whose function is to secrete mucus. This nasal mucous membrane is firmly blended and adherent to the underlying respective cartilaginous or bony structures of the nose.

Hilding *(82)* states that there is no structure in the body so delicate and so severely exposed to the environment as the nasal mucosa. "One would expect an inert, resistent, leathery type of epithelium in such an exposed position. Instead there is a highly specialized, ciliated, fragile membrane. It exists largely by virtue of its remarkable active system of defense."

He explains that the epithelium of the upper part of the respiratory tract is not a static structure, but that the cilia beat with extreme rapidity and move tremendous loads of mucin and foreign material with speed, power and efficiency. It may be said that there is a complete new lining of mucus over all the ciliated areas every ten or fifteen minutes. Even the nonciliated, inactive areas, in the anterior third of the nose have an exchange of mucin every hour or so. Drainage of mucin from the inactive area is accomplished by traction on the threads of mucin in the secretion.

Hilding continues to explain that when an irritant attacks the surface, the mucosa sloughs its injured cells readily before they are dead, and replaces them very quickly. Prolific regeneration seems to be part of the physiologic process. However, when exposed to excessive amounts of irritants, the epithelium of the membrane is altered in form from the active, ciliated type to the more inactive squamous type. As a result of this the cleansing action of this membrane is greatly reduced.

Beck *(83)* studied the effect of chromic acid on the nasal mucous membrane by taking portions of the turbinates for study at intervals of one hour to fourteen days. He concluded from this study that there was fixation of the tissue by coagulation of the proteins.

In 1930, Lukanin *(25)* exposed rabbits in various departments of the Ural Government Chromate Works (USSR). He was able to produce nasal septal perforation in a rabbit after four month's exposure in the section of the plant where sodium dichromate was being evaporated.

Dixon *(33)* in a detailed study of 18 cases of workers with septal perforation stated that the ulcers usually begin about ¼ inch posterior to and above the anterior-inferior edge of the septum, then progress upward and backward. This site is known as Kiesselbach's or Little's area.

The following are anatomical predisposing factors for the ulceration and perforation to take place at this site:

1. This area is in line with the sweep of dust and vapors taken in through inhalation.
2. This area lacks cilia, so that mucin is propelled very slowly.

3. This area is the transition of very thin modified stratified squamous epithelium to that of ciliated epithelium.
4. The firm adherence of the mucous membrane to the underlying cartilage enhances the involvement of this cartilage.

The following are some of the physiologic and pathologic considerations involved in the formation of the nasal lesion:

1. The filtering function of the nose cannot handle the amount and type of exposure.
2. Both dry and hot conditions that often exist in these industrial environments will interfere with the normal protective mechanism of the mucin-moving blanket.
3. The corrosive action of the chromates, and the marked change of pH that it usually produces, seriously interfere with the protective action the mucin and cilia would normally afford.
4. The protein coagulation action of chromates and bichromates, which has long been known, specifically necrotizes the nasal mucosa.

In observing the progressive pathological changes of this lesion one first notices that the entire nasal mucous membrane is markedly inflamed and dry. Then an area of necrosis develops just posterior to Little's area on both sides of the septum. Crusts of dried mucin and foreign material tend to develop at these sites. Upon the removal of the crusts (blowing or picking) the necrotic epithelial layers come off with them, leaving behind fresh ulcers which in turn become dirty and gray, and always remain dry.

Immediately surrounding each ulcer is a narrow zone of inflammatory reaction which in turn is surrounded by a zone of hyperplasia and injection of blood vessels, which blends into the rest of the more mildly inflamed nasal mucous membrane. The ulcers constantly increase in size and depth with repeated crust formation and removal until eventually they erode through the septum resulting in a perforation. This usually occurs upon the removal of a crust which includes, besides dried mucin, the last necrotic tissue between the two ulcers. The perforation continues to increase in size with the formation of larger crusts, which are called "clinkers" or "scabs" by the workmen.

The perforation increases in size, proceeding superiorly and posteriorly until it reaches the vomer and the perpendicular plate of the ethmoid. In none of the previous reports nor in our survey were the osseous or cartilaginous structures involved. The shape of the nose was never affected. In the old perforations, where the entire nasal septal cartilage was destroyed, the margins of the ulcer revealed thick, well-healed borders and did not have the hypersensitive zone which is present in the progressively enlarging perforation and also in the ulceration. However, even in the larger perforations scab formation (clinkers) still takes place, increasing in size with the perforation.

Although this lesion is frequently found in chromate workers with bronchiogenic carcinoma, no direct correlation has been established. In none of the reports in the literature has any neoplasm developed in the site of the perforation. However, Newman *(84)* reports a neoplasm, attached to the inferior turbinate, occurring in a chromate worker who had a nasal perforation. This neoplasm on section was found to be an adenocarcinoma, but did not involve the site of the perforation.

*Findings.*—In Table 28, the status of the nasal mucous membrane of 897 chromate workers is tabulated by color and years in the chromate industry. Of these workers, 509 had nasal septal perforation, a prevalence of 56.7 percent. Of the 653 white chromate workers, 322 (49.3 percent) had perforations, whereas among the 244 colored chromate workers 187, or 76.6 percent, had perforations. Thus, the colored chromate workers show a greater prevalence of nasal perforations than do the white chromate workers.

When examined at the time of this survey none of the white workers who had been employed less than six months had a nasal perforation but 11.1 percent of the colored workers were so affected. With six months to three years of chromate experience 31.5 percent of the white workers and 64.3 percent of the colored workers had developed nasal perforation. All workers did not eventually show a perforation. Among the white males examined there were 107 workers (36 percent) with 10 years or over in the chromate industry who failed to show perforation. Among colored workers with a similar experience there were five individuals (6.9 percent) who did not show perforation.

TABLE 28.—*Percent of chromate workers with perforation of nasal septum, according to time worked in the industry, by color.*

| Time worked in chromate industry | Both colors | | | White | | | Colored | | |
|---|---|---|---|---|---|---|---|---|---|
| | Total workers | Number with perforation | Percent with perforation | Total workers | Number with perforation | Percent with perforation | Total workers | Number with perforation | Percent with perforation |
| Total.. | 897 | 509 | 56.7 | 653 | 322 | 49.3 | 244 | 187 | 76.6 |
| Less than 6 months. | 41 | 1 | 2.4 | 32 | 0 | 0 | 9 | 1 | 11.1 |
| 6 months–3 years. | 117 | 46 | 39.3 | 89 | 28 | 31.5 | 28 | 18 | 64.3 |
| 3–10 years. . | 370 | 205 | 55 4 | 235 | 104 | 44.3 | 135 | 101 | 74.8 |
| 10 years or over | 369 | 257 | 69.6 | 297 | 190 | 64.0 | 72 | 67 | 93.1 |

In the nonchromate groups, both white and colored, only one man had perforation of the nasal septum. This man proved to be one who had worked previously in a chromate-producing plant, at which time he had obtained his perforation.

The most common nose finding in the chromate workers was crusting, which was present in about two-thirds of all groups.

In Table 29 the duration of employment in the chromate industry is correlated with the onset of nasal perforation. It is noticed that 42.3 percent of

the white chromate workers who acquired nasal perforation experienced it within the first year, compared to 65.7 percent of the colored chromate workers. However, lesions acquired the first 6 months were nearly the same, 23.0 for white and 24.1 percent for colored workers. After working seven years in chromate exposure, 86.4 percent of the white and 99.4 percent of the colored workers with perforation had experienced their lesion. These percentages obviously refer to conditions which prevailed in the past and workers entering the chromate industry at the present time might not acquire nasal perforation so rapidly.

TABLE 29.—*Distribution of chromate workers with perforated nasal septum according to time worked in industry before getting perforation, by color.*

| Time in chromate industry before getting perforated septum | Total | White | Colored | Total | White | Colored |
|---|---|---|---|---|---|---|
| | Number | | | Percent | | |
| Total | 473 | 295 | 178 | 100.0 | 100.0 | 100.0 |
| Less than 6 months | 111 | 68 | 43 | 23.5 | 23.0 | 24.1 |
| 6–12 months | 131 | 57 | 74 | 27.7 | 19.3 | 41.6 |
| 1–3 years | 96 | 64 | 32 | 20.3 | 21.7 | 18.0 |
| 3–7 years | 94 | 66 | 28 | 19.9 | 22.4 | 15.7 |
| 7 years or over | 41 | 40 | 1 | 8.6 | 13.6 | .6 |

The length of time from the beginning of exposure until perforation occurs has been reported as varying widely. A report by the medical inspector of factories in England *(5)* in 1930 gives the range as from 6 months to 4 years. Legge *(4)* noted one case as early as 7 weeks, but agreed that generally perforation occurred from 6 to 12 months after employment. Carter *(32)* reported a latent period of from 2 to 3 months after the initial exposure. Dixon *(33)*, in his series of 18 cases, found perforations of one-quarter of an inch in diameter in two employees who had worked only 6 weeks at the plant. Probably the shortest periods of exposure for the development of this condition reported in the literature are those noted by Delpech and Hillairet *(3)* and Fischer [cited by Thompson *(13)*] each of whom had examined a worker who had developed septal perforation after only one week of exposure. These men were both employed in bichromate-producing plants.

It is recorded that some exposed individuals do not get perforations even though exposure may be to high concentrations of chromates. Of the 30 men examined by Legge *(4)*, and who had no perforations, over half had worked more than 10 years, and many had worked in relatively high exposure areas. Legge, and Zvaifler *(85)* feel that there are individuals whose nasal mucous membranes are more resistant than others to the action of the chromates.

In correlating the number of baths per week and the prevalence of nasal perforation, no definite relationship could be established.

No correlation was found between smoking and the prevalence of nasal perforation in chromate workers. Among the white workers 20.8 percent with perforation and 19.1 percent without perforation did not smoke. The corresponding percentages among colored workers who did not smoke were 14.4 and 21.1.

White and colored chromate workers revealed no significant relationship between frequency of common colds during the past year and presence or absence of nasal perforation.

A detailed study was made of 87 chromate workers in regard to the practice of different nasal prophylactic measures and the occurrence of ulceration and perforation. Of the total workers 50.6 percent used some type of prophylaxis. All of the six men with no ulceration and 75.7 percent of the 37 men with early or small ulceration, used some prophylactic measures. However, some means of prophylaxis was used by only 19.5 percent of the 41 men with perforation, but by 78.3 percent of the 46 men without perforation. This suggests a relationship between the absence of perforation and the practice of prophylaxis.

In evaluating the relative benefits of different types of prophylaxis, namely, mask, petrolatum, or nasal douching, it was observed that a combination of all three types of prophylaxis was more often associated with the absence of nasal ulceration or perforation than was any one method used alone.

Figures 8 and 9 present examples of nasal perforation encountered in this study.

### *Throat*

The presence of a severely red throat was found in 9.8 percent of the white chromate workers, and in 12.8 percent of the colored chromate workers, but in only 1.4 percent of the white nonchromate workers and 7.4 percent of the colored nonchromate workers. From Table 30 it will be observed that the presence of edema of the uvula was found in 5.7 percent of the white chromate workers and in 12.3 percent of the colored chromate workers, compared with 1.4 percent and 4.9 percent for nonchromate workers. However, the presence of pharyngeal discharge was not appreciably different in the chromate and nonchromate workers but was greater among the white workers. Pharyngeal or soft palate ulcerations were not found as frequently as has been reported in the literature *(12, 32, 36, 37, 38)*.

Bloomfield and Blum *(31)* report that a concentration of chromic acid beyond 0.1 mg per cubic meter of air is sufficient to injure the nasal mucous membrane. However, Legge *(4)* did not find definite ulceration of the throat in his study, but did note the presence of white mucous patches on the pharynx. Mancioli *(39)* called attention to chronic pharyngitis along with ulceration of the larynx and of the vocal cords in Italian bichromate

workers. Wilensky *(23)* found that 41.7 percent of 278 bichromate workers whom he examined had pharyngitis.

Lieberman *(38)* described the throat of chromate workers as being dry, red, and having a glazed appearance. He also mentions the presence of a whitish pseudo-membrane, which appeared on the posterior surface of the tongue and the floor of the mouth of two of the five cases examined. The tongues of these two were coated, dry, and cracked, with many of the papillae enlarged and red. One of the men complained of impairment of taste.

TABLE 30.—*Selected throat findings and symptom among chromate and nonchromate workers, by color.*

| Throat findings and symptom | Number | | | Percent | | |
|---|---|---|---|---|---|---|
| | Total | White | Colored | Total | White | Colored |
| | Chromate | | | | | |
| Total workers | 897 | 653 | 244 | ........ | ........ | ........ |
| *Findings:* | | | | | | |
| Edema of uvula | 67 | 37 | 30 | 7.5 | 5.7 | 12.3 |
| Discharge on pharyngeal wall | 49 | 40 | 9 | 5.5 | 6.1 | 3.7 |
| Severely reddened | 95 | 64 | 31 | 10.6 | 9.8 | 12.8 |
| *Symptom:* | | | | | | |
| Hoarseness | 53 | 43 | 10 | 5.9 | 6.6 | 4.1 |
| | Nonchromate | | | | | |
| Total workers | 155 | 74 | 81 | ........ | ........ | ........ |
| *Findings:* | | | | | | |
| Edema of uvula | 5 | 1 | 4 | 3.2 | 1.4 | 4.9 |
| Discharge on pharyngeal wall | 5 | 4 | 1 | 3.2 | 5.4 | 1.2 |
| Severely reddened | 7 | 1 | 6 | 4.5 | 1.4 | 7.4 |
| *Symptom:* | | | | | | |
| Hoarseness | 12 | 7 | 5 | 7.7 | 9.5 | 6.2 |

A clinical and X-ray examination of the oronasal and laryngeal structures was done recently on 97 workers in a chromate-producing plant in Ohio, and reported by Mancuso *(34)*. In this group, 86.6 percent had chronic chemical rhinitis, 42.3 percent chronic chemical pharyngitis; laryngitis with hoarseness occurred in 10.3 percent and congestion of the vocal cords without hoarseness in another 10.3 percent. Thickened sinus membranes were present in 27.0 percent of the workers, and there was a total incidence of polyps of these structures of 11.8 percent, with the majority occurring in the sinuses. In 10.3 percent it was observed that there was a loss or reduction in the sense of taste.

### *Ears*

Examination of the ears revealed thickened drums to be more prevalent among white workers than among colored, in both chromate and nonchromate workers. Table 31 shows that no marked difference existed between the chromate and nonchromate workers. Perforation and discharge were

found to be slightly less prevalent in the white chromate workers than in the nonchromate workers.

TABLE 31.—*Selected ear findings and symptoms among chromate and nonchromate workers, by color.*

| Ear findings and symptoms | Number | | | Percent | | |
|---|---|---|---|---|---|---|
| | Total | White | Colored | Total | White | Colored |
| | Chromate | | | | | |
| Total workers | 897 | 653 | 244 | ........ | ........ | ........ |
| *Findings:* | | | | | | |
| Thickened drum | 165 | 135 | 30 | 22.5 | 25.7 | 14.4 |
| Perforated drum | 18 | 15 | 3 | 2.4 | 2.9 | 1.4 |
| Discharge | 8 | 8 | 0 | 1.1 | 1.5 | .... |
| Any finding | 249 | 191 | 58 | 33.9 | 36.3 | 27.8 |
| *Symptoms:* | | | | | | |
| Impaired hearing | 82 | 64 | 18 | 9.3 | 10.0 | 7.5 |
| Tinnitus | 45 | 31 | 14 | 3.1 | 4.8 | 5.8 |
| Any symptom | 124 | 96 | 28 | 14.1 | 15.0 | 11.7 |
| | Nonchromate | | | | | |
| Total workers | 155 | 74 | 81 | ........ | ........ | ........ |
| *Findings:* | | | | | | |
| Thickened drum | 32 | 17 | 15 | 22.9 | 25.4 | 20.5 |
| Perforated drum | 3 | 2 | 1 | 2.1 | 3.0 | 1.4 |
| Discharge | 2 | 2 | 0 | 1.4 | 3.0 | .... |
| Any finding | 51 | 26 | 25 | 36.4 | 38.8 | 34.2 |
| *Symptoms:* | | | | | | |
| Impaired hearing | 6 | 3 | 3 | 3.9 | 4.1 | 3.7 |
| Tinnitus | 4 | 1 | 3 | 2.6 | 1.4 | 3.7 |
| Any symptom | 10 | 4 | 6 | 6.5 | 5.4 | 7.4 |

NOTE.—Percentages based on workers for whom data were available.

Impaired hearing (subjective findings) was noted in 10.0 percent of the white and 7.5 percent of the colored chromate workers as compared to 4.1 percent and 3.7 percent in the white and colored nonchromate groups, respectively. In the examination of 97 workers in a chromate-producing plant, Mancuso *(34)* found 68.8 percent had some hearing loss, according to audiometric tests.

## Dental findings

It has been reported that the teeth of workers in chromate-producing plants become stained a yellow color *(86)*. Inflammation and ulceration of the mucous membrane are said to develop as a result of exposure to chromates *(87)*. The tongue, in some cases, may be sore with many of the papillae red and enlarged *(38)*.

### *Methods and population studied*

An inspection-type of dental examination was conducted by two dentists and performed at the time the workers reported for a physical examination. No previous instructions were given relative to brushing the teeth prior to the visit. The patient was seated in a portable dental chair with a diagnostic light properly adjusted to assure good illumination. Sufficient time was

permitted to inspect the teeth with a plane mouth mirror and explorer. All pathologic conditions and abnormalities of the lips, mucous membranes, gingivae, teeth, tongue, palate, uvula, and velum were noted and recorded.

A total of 561 chromate workers was given a dental examination of which number 365 were white and 196 colored. They ranged in age from 17 to 73 years for the white and 18 to 66 years for the colored, with a median age of 40.9 and 42.0 years, respectively. Generally speaking, the white workers examined were of a heterogeneous origin with 32.1 percent foreign born, mostly middle European, while the remaining 67.9 percent were native born whites from the Atlantic seaboard states.

For purposes of comparison a similar group of workers who were not exposed to chromates were examined, of which 66 were white and 58 colored.

### *Dental caries*

The amount of dental caries experience observed in the permanent teeth of a particular age group may be expressed in terms of the number of teeth with untreated dental caries, the number of extracted teeth (including teeth indicated for extraction), and the number of teeth filled. All three findings (decayed, missing, or filled) may be considered separately or may be combined into a single rate known as the DMF rate. In calculating this rate each tooth is given one of the above designations. A tooth containing both a filled and one or more carious lesions is counted as a filled tooth. All teeth indicated for extraction are considered as missing teeth. Thus, the same tooth may not be counted more than once. The unit of measurement is the individual tooth, not the tooth surface. In each mouth the total number of teeth given consideration is 28, the four third molars being omitted.

TABLE 32.—*Rate per person of decayed, missing and filled teeth (DMF) among 561 chromate and 124 nonchromate workers, according to age, by color.*

| Age group (years) | White | | | | | | Colored | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Number of workers | Number edentulous | Rate per person | | | | Number of workers | Number edentulous | Rate per person | | | |
| | | | D | M | F | DMF | | | D | M | F | DMF |
| | Chromate | | | | | | | | | | | |
| Total | 365 | 45 | 1.1 | 12.0 | 3.6 | 16.7 | 196 | 10 | 1.1 | 7.9 | 1.0 | 10.0 |
| Under 35 | 113 | 3 | 1.6 | 6.2 | 6.4 | 14.2 | 53 | 0 | 1.3 | 3.0 | 1.8 | 6.1 |
| 35–44 | 117 | 11 | 1.0 | 12.3 | 3.7 | 17.0 | 60 | 0 | 1.3 | 5.9 | 1.1 | 8.3 |
| 45 or over. | 135 | 31 | .7 | 16.6 | 1.2 | 18.5 | 83 | 10 | .9 | 12.6 | .5 | 14.0 |
| | Nonchromate | | | | | | | | | | | |
| Total | 66 | 7 | 1.6 | 9.5 | 6.1 | 17.2 | 58 | 1 | 1.6 | 5.9 | .8 | 8.3 |
| Under 35 | 30 | 2 | 1.8 | 6.6 | 7.4 | 15.8 | 30 | 0 | 2.1 | 5.4 | 1.0 | 8.5 |
| 35–44 | 24 | 1 | 1.8 | 8.6 | 6.3 | 16.7 | 9 | 0 | 1.7 | 3.8 | .6 | 6.1 |
| 45 or over | 12 | 4 | .4 | 18.5 | 2.4 | 21.3 | 19 | 1 | .7 | 7.7 | .7 | 9.1 |

NOTE.—DMF based on 28 teeth per person, edentulous excluded.

Table 32, in addition to other items, shows the DMF rate for the white and colored chromate and nonchromate workers according to specific age

groups. It will be observed that the total DMF for all ages of the white workers is 16.7 per chromate worker and 17.2 per nonchromate worker, and for the colored chromate worker it is 10.1 as compared to 8.3 for the colored nonchromate worker. These differences between the chromate and nonchromate workers are not statistically significant. When consideration is given to the specific age groups the differences again are not significant. There is no recognizable evidence, therefore, to indicate that exposure to chrome compounds affected the rate of caries attack for either the white or colored workers.

The difference in the respective components of the DMF may be an indication of the extent to which the problem of dental caries has been met. A carious tooth when left untreated eventually becomes a tooth indicated for extraction or a missing one. It then follows that the more carious teeth that have been filled the fewer will be the teeth indicated for extraction or missing.

It is of interest to note that there appears a difference in the respective components of the DMF when the data for white chromate workers are compared with those for white nonchromate workers. Of the total DMF for the white chromate workers, the decayed teeth represent 6.3 percent; the missing teeth, 72.0 percent; the filled teeth, 21.7 percent; while for the white nonchromate workers these corresponding percentages are 9.1, 55.3, and 35.6 percent. These percentages are shown in the accompanying tabulation.

| Workers | Percentage, composition of DMF | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | Total | Decayed | Missing | Filled | Total | Decayed | Missing | Filled |
| | White | | | | Colored | | | |
| Chromate.... | 100 | 6.3 | 72.0 | 21.7 | 100 | 11.2 | 78.6 | 10.2 |
| Nonchromate | 100 | 9.1 | 55.3 | 35.6 | 100 | 19.0 | 71.2 | 9.8 |

Since the total DMF, or the rate of caries attack, for the two groups is about the same, it is evident that the nonchromate workers gave more attention to the treatment of caries than did the chromate workers, with the result that they have more teeth filled and fewer teeth missing.

### *Keratosis and inflammation*

It will be seen from table 33 that among the chromate workers the percent having inflammation of the specified structures is generally higher than for the nonchromate workers. Keratosis is considered a normal sequel to an inflammatory process and since this latter condition is present among a higher percent of chromate workers, it is not surprising to find more keratosis among them also.

### *Yellow teeth and tongue*

During the course of the study it was noted that 32.8 percent of the white chromate workers and 48.9 percent of the colored chromate workers had a yellowish discoloration of the anterior teeth, and 45.8 percent of the white

TABLE 33.—*Number and percent of chromate and nonchromate workers showing selected abnormalities of oral structures, by color.*

| Abnormality | White | | Colored | |
|---|---|---|---|---|
| | Chromate | Nonchromate | Chromate | Nonchromate |
| | Number | | | |
| Total population | 365 | 66 | 196 | 58 |
| Total population minus edentulous | 320 | 59 | 186 | 57 |
| Inflammation: | | | | |
| Velum | 59 | 5 | 2 | 0 |
| Uvula | 170 | 27 | 56 | 16 |
| Palate | 44 | 6 | 3 | 1 |
| Keratosis: | | | | |
| Lips | 42 | 2 | 1 | 1 |
| Gingiva | 57 | 2 | 15 | 1 |
| Palate | 79 | 11 | 15 | 5 |
| Yellow stained teeth | 105 | 0 | 91 | 2 |
| Yellow stained tongue | 167 | 9 | 74[1] | 2 |
| Gingivitis | 239 | 33[1] | 156 | 26 |
| Periodontitis | 223[2] | 20[3] | 135 | 23 |
| | Percent | | | |
| Inflammation: | | | | |
| Velum | 16.2 | 7.6 | 1.0 | 0 |
| Uvula | 46.6 | 40.9 | 28.6 | 27.6 |
| Palate | 12.1 | 9.1 | 1.5 | 1.7 |
| Keratosis: | | | | |
| Lips | 11.5 | 3.0 | .5 | 1.7 |
| Gingiva [4] | 17.8 | 3.0 | 8.1 | 1.7 |
| Palate | 21.6 | 16.7 | 7.7 | 8.6 |
| Yellow stained teeth [4] | 32.8 | 0 | 48.9 | 3.5 |
| Yellow stained tongue | 45.8 | 13.6 | 37.9 | 3.4 |
| Gingivitis [4] | 74.7 | 56.9 | 83.9 | 45.6 |
| Periodontitis [4] | 70.3 | 35.1 | 72.6 | 40.4 |

[1] One person with data not stated.
[2] Three persons with data not stated.
[3] Two persons with data not stated.
[4] Only persons with teeth considered.

and 37.9 percent of the colored chromate workers had a yellowish coating of the dorsum of the tongue. The color ranged from a light yellow to a dark brown.

For the purpose of this presentation the discolorations from light yellow to brown are grouped and referred to as a yellow discoloration. The teeth most often affected were the upper and lower anterior incisors. As is seen in figure 10, the labial surfaces of the anterior teeth were involved. In some cases the discoloration showed an elliptical pattern, the periphery of which paralleled the upper and lower lip line. The discoloration was superficial since it was easily removed with ordinary dental scaling and prophylactic instruments.

A similar discoloration was observed on the anterior two-thirds of the tongue. See figure 11. The colors appeared to vary from yellow to brown, depending upon the anatomy of the tongue.

Persons with a tongue appearing to be smooth and apparently denuded presented no discoloration of the tongue. Differences in the occurrence of a yellowish discoloration of the anterior teeth and the dorsum of the tongue may not be attributed to the use of tobacco since the proportion of workers using tobacco was found to be similar for both the chromate and nonchromate groups. Scrapings were obtained and chemically analyzed from

a large number of workers with a yellow tongue. In addition to other elements such as calcium, magnesium, copper and manganese, a small amount of chromium was recovered.

The level of chromate exposure necessary to bring about a discoloration of the tongue apparently is very low since persons working long distances from the processing of the chromates developed a yellow tongue. In one plant studied, personnel regularly employed in the air-conditioned dispensary developed the characteristic yellow discoloration of the anterior two-thirds of the tongue similar to that observed among the workers in the refining process. Likewise persons participating in the conduct of the examinations of this study, which were done in the same dispensary, also experienced a slight yellow discoloration of the tongue. Associated with the onset of the discoloration, there was a certain amount of dryness of the mouth accompanied by a metallic taste. This latter experience disappeared after the second or third day.

### *Periodontal diseases*

The periodontal diseases considered are gingivitis and periodontitis without reference to severity. The former is an inflammation of the gingival tissue while the latter is an inflammatory condition involving degenerative changes of the alveolar bone accompanied by a recession of the gingival tissue. Data for periodontitis and periodontosis are grouped and referred to as periodontitis.

As seen in Table 33, the percent of persons affected with gingivitis and the percent of persons affected with periodontitis are significantly higher among the chromate workers for both races.

Since age is an influencing factor, table 34 shows in number and percent the prevalence of periodontal disease among chromate and nonchromate workers according to specific age groups. For white chromate workers of all ages, 74.7 percent of the total workers had gingivitis, as compared to 56.9 percent for the white nonchromate workers. For the colored workers, these corresponding figures are 83.9 percent for chromate workers, and 45.6 percent for nonchromate workers. When consideration is given to each age group, the chromate workers generally show a higher percentage with gingivitis than do the nonchromate workers for both races.

The prevalence of periodontitis, like gingivitis, was observed to be higher among the chromate workers. There is a gradual increase in the percent of persons affected with advance in age for these two industrial groups studied, although at different levels. As stated by Bernier *(88)*, recession of the gingiva may occur as a normal physiological process associated with aging. In this change the process of inflammation plays a definite role, being the exciting mechanism which is responsible for the clinical change.

The percent of white chromate workers of all ages with periodontitis is 70.3 as compared with 35.1 percent for white nonchromate workers. The corresponding percentages for the colored workers are 72.6 for the chromate,

TABLE 34.—*Number and percent of chromate and nonchromate workers showing gingivitis and periodontitis, by age and color.*

| Worker group | Gingivitis: Total | Gingivitis: Age (years) Under 35 | Gingivitis: 35–44 | Gingivitis: 45 or over | Periodontitis: Total | Periodontitis: Age (years) Under 35 | Periodontitis: 35–44 | Periodontitis: 45 or over |
|---|---|---|---|---|---|---|---|---|
| | Number with specified findings | | | | | | | |
| | White | | | | | | | |
| Chromate.... | 239 | 75 | 79 | 85 | 223 [1] | 46 | 81 | 96 |
| Nonchromate | 33 | 12 | 14 | 7 | 20 [2] | 3 | 10 | 7 |
| | Colored | | | | | | | |
| Chromate.... | 156 | 43 | 51 | 62 | 135 | 24 | 46 | 65 |
| Nonchromate | 26 | 12 | 7 | 7 | 23 | 5 | 6 | 12 |
| | Percent of total workers [3] | | | | | | | |
| | White | | | | | | | |
| Chromate... | 74.7 | 68.2 | 74.5 | 81.7 | 70.3 | 42.2 | 76.4 | 94.1 |
| Nonchromate | 56.9 [4] | 44.4 | 60.9 | 87.5 | 35.1 | 11.1 | 45.5 | 87.5 |
| | Colored | | | | | | | |
| Chromate.... | 83.9 | 81.1 | 85.0 | 84.9 | 72.6 | 45.3 | 76.7 | 89.0 |
| Nonchromate | 45.6 | 40.0 | 77.8 | 38.9 | 40.4 | 16.7 | 66.7 | 66.7 |

[1] Three with data not stated.
[2] Two with data not stated.
[3] Percentages based on total number with teeth.
[4] One with data not stated.

and 40.4 for the nonchromate workers. Similarly, for each specific age group the chromate workers show a higher percentage with periodontitis. With respect to the prevalence of periodontitis, the colored workers follow the same general pattern as the white workers and at about the same level for the corresponding industrial groups, thus showing no race differences.

It is reported that periodontal diseases may be the result of many complex influences such as general and local metabolic disturbances, infection, and inflammation resulting from local irritating agents *(89)*. In many cases, a combination of the foregoing factors may be responsible. No effort was made therefore to assess specifically the extent environmental irritating agents contributed to the development of the periodontal diseases observed.

## Chest

### *Lungs*

The pulmonary findings in this portion of the medical report include all aspects other than neoplastic growths. The latter subject will be reviewed in a separate section later in this report.

*Vital capacity.*—Vital capacity studies were made with the realization that limitations exist in this estimation of pulmonary function *(90)*. Comparisons, however, with former surveys *(79)* conducted by this division in which

a definite high prevalence of pulmonary fibrosis was found, are of some interest. The vital capacity records were used as actual readings, rather than making calculations as to their percentage of normal.

Median vital capacity readings for chromate and nonchromate workers show practically no difference, the reading for white workers being slightly higher in both groups. Since vital capacity decreases with age, as shown in Table 35, and the white chromate workers are on an average three years older than the colored chromate workers, a color difference is observed.

Comparing chromate workers with ferrous foundry workers *(79)* it is found that the median vital capacity is a little lower for the foundry workers although they are, on an average, younger than the chromate workers.

TABLE 35.—*Vital capacity of chromate and nonchromate workers, by age and color.*

| Vital capacity (liters) | Chromate | | | | Nonchromate | | | |
|---|---|---|---|---|---|---|---|---|
| | Total | Age (years) | | | Total | Age (years) | | |
| | | Under 40 | 40–49 | 50 or over | | Under 40 | 40–49 | 50 or over |
| | Number of white workers | | | | | | | |
| Total | 648 | 258 | 137 | 253 | 71 | 51 | 11 | 9 |
| Less than 3.0 | 55 | 1 | 5 | 49 | 4 | 1 | 1 | 2 |
| 3.0–3.9 | 253 | 66 | 55 | 132 | 25 | 14 | 5 | 6 |
| 4.0–4.9 | 283 | 151 | 67 | 65 | 32 | 27 | 4 | 1 |
| 5.0 or over | 57 | 40 | 10 | 7 | 10 | 9 | 1 | 0 |
| Median | 4.1 | 4.4 | 4.1 | 3.6 | 4.2 | 4.2 | 3.9 | 3.5 |
| | Number of colored workers | | | | | | | |
| Total | 221 | 110 | 65 | 46 | 79 | 49 | 14 | 16 |
| Less than 3.0 | 24 | 8 | 9 | 7 | 12 | 4 | 3 | 5 |
| 3.0–3.9 | 123 | 52 | 41 | 30 | 38 | 24 | 6 | 8 |
| 4.0–4.9 | 69 | 49 | 12 | 8 | 27 | 20 | 5 | 2 |
| 5.0 or over | 5 | 1 | 3 | 1 | 2 | 1 | 0 | 1 |
| Median | 3.7 | 3.7 | 3.6 | 3.5 | 3.7 | 3.9 | 3.7 | 3.4 |
| | Percent of white workers | | | | | | | |
| Total | 100.0 | 100.0 | 100.0 | 100.0 | 100.0 | 100.0 | 100.0 | 100.0 |
| Less than 3.0 | 8.5 | 0.4 | 3.7 | 19.3 | 5.6 | 2.0 | 9.1 | 22.2 |
| 3.0–3.9 | 39.0 | 25.6 | 40.1 | 52.2 | 35.2 | 27.4 | 45.4 | 66.7 |
| 4.0–4.9 | 43.7 | 58.5 | 48.9 | 25.7 | 45.1 | 52.9 | 36.4 | 11.1 |
| 5.0 or over | 8.8 | 15.5 | 7.3 | 2.8 | 14.1 | 17.7 | 9.1 | 0 |
| | Percent of colored workers | | | | | | | |
| Total | 100.0 | 100.0 | 100.0 | 100.0 | 100.0 | 100.0 | 100.0 | 100.0 |
| Less than 3.0 | 10.8 | 7.3 | 13.8 | 15.2 | 15.2 | 8 2 | 21.4 | 31.3 |
| 3.0–3.9 | 55.7 | 47.3 | 63.1 | 65.2 | 48 1 | 49.0 | 42.9 | 50.0 |
| 4.0–4.9 | 31.2 | 44.5 | 18.5 | 17.4 | 34.2 | 40.8 | 35.7 | 12.5 |
| 5.0 or over | 2.3 | .9 | 4.6 | 2.2 | 2.5 | 2.0 | 0 | 6.2 |

*Pulmonary fibrosis.*—In evaluating the effect on the lungs of the various contaminants in the working environment, chest roentgenograms of 897 chromate workers were classified according to intensity of linear fibrosis, appearance of graininess (ground glass) and nodulation. Generally, films classified as normal, linear exaggeration one, and linear exaggeration two

may be considered as being within normal limits or due to lung changes unrelated to the dustiness of the occupational environment. The ground glass one appearance may or may not be due to the dust in the environment. However, films showing ground glass two, generalized nodulation and conglomerate masses of nodules (with other medical findings essentially negative) are strongly suggestive of an effect due to dust of a past occupational environment.

Letterer *(43)*, and Letterer and co-workers *(44)* reported finding interstitial and interalveolar fibrotic processes on autopsying chromate dye workers. Mancuso *(34)*, and Mancuso and Hueper *(78)* on histologic examination found this phenomenon in the lungs of three former workers of a chromate-producing plant. They referred to it as "chromitotic pneumoconiosis." The chief abnormality in the chest X-rays of these workers was slight bilateral hilar enlargement.

It was found that in the chromate workers 98 persons manifested normal lung markings, 376 showed linear one markings, 323 showed linear two markings, 44 showed ground glass one, 11 persons or 1.3 percent of all chromate workers had ground glass two, and only one worker showed nodular markings. (This particular worker, a 39-year-old man, had been a welder in both present and previous occupations.)

The pulmonary markings increased with age in accordance with the findings of previous studies. No correlation with occupation in the chromate-producing industry could be made among the chromate workers with ground glass two pulmonary markings. Their past occupations did not suggest exposure to silica dust. Thus on the basis of X-ray data we cannot confirm the presence of pneumoconiosis from chromate exposure.

The prevalence of pulmonary markings suggestive of fibrosis in the nonchromate workers was even less significant than in the chromate workers. However, it was noted that bilateral hilar enlargements were seen in the chest films of chromate workers. This is consistent with the findings of Letterer *(43)*, Letterer and co-workers *(44)*, Mancuso *(34)*, and Mancuso and Hueper *(78)*.

*Pulmonary tuberculosis.*—The criteria for interpreting the stage of reinfection type of pulmonary tuberculosis in this study were those of the National Tuberculosis Association *(91)*.

The degree of activity of the suspected tuberculosis cases was classified inactive or of doubtful activity, dependent upon the general characteristics of the roentgenographic shadows as well as upon clinical data. Pulmonary tuberculosis was suspected from the roentgenograms of ten chromate workers, with an almost identical percentage in both white and colored groups; 1.1 percent and 1.3 percent, respectively. The nonchromate group revealed only one case of tuberculosis, a colored worker with questionable minimal tuberculosis, inactive. Tuberculosis in the ten chromate workers with suspicious roentgenograms was classified as minimal, probably inactive.

Observing findings of a tuberculosis survey carried out by the Denver Tri-County Chest Survey *(92)* in 1949, including some 324,000 people, we find the rate per 10,000 persons for active, far-advanced tuberculosis to be 3; for active, moderately advanced to be 5; for active, minimal to be 2; for questionably active, far advanced to be 1; for questionably active, moderately advanced to be 8; and for questionably active, minimal to be 7; for inactive, far advanced to be 5 and for inactive moderately advanced to be 31; as compared to no instances among the chromate workers. However, the rate for inactive, minimal tuberculosis was 65 per 10,000 in the Denver survey as compared to 110 per 10,000 among chromate workers. Thus, one can note that the severity of tuberculosis is less among the chromate workers.

One would anticipate an apparent low prevalence of tuberculosis in both the chromate and nonchromate groups as compared to the prevalence in routine mass screening surveys. In one such survey a rate of 11.6 cases of active tuberculosis per 10,000 men of similar age is reported *(93)*. The chromate plants have a periodic (six months to a year) X-ray examination of the chest for all their employees. This, over a period of time, would tend to decrease the number of active cases of tuberculosis that may be found in this group of employees as compared to others where such a program is not in operation.

### *Cardiovascular findings*

*Criteria and method.*—The criteria for interpreting symptoms and signs and diagnosis for heart disease were those of the New York Heart Association *(94)*.

Parts of the general physical examination with reference to the cardiovascular system included the palpation and auscultation of the heart, blood serological test for syphilis, and chest roentgenogram taken at 6 foot target distance. The lack of an electrocardiogram, as well as the inability to study the worker on more than one occasion, is acknowledged.

In all the chromate-producing plants as well as in the plants where nonchromate groups were selected, preplacement examinations which included evaluation of the cardiovascular system were required. Therefore the prevalence of cardiovascular diseases in these groups would be anticipated to be lower than that found in a similar age group of male workers who do not undergo such a screening procedure.

*Heart disease.*—Of a total of 653 white chromate workers 21, or 3.2 percent, were diagnosed as having heart disease. Nine had hypertensive heart disease, eight had arteriosclerotic heart disease, three had rheumatic heart disease and one had syphilitic heart disease. In the 244 colored chromate workers 9, or 3.7 percent, were diagnosed as having heart disease. Five had hypertensive heart disease, three had syphilitic heart disease and one had rheumatic heart disease. The median age of the 21 white chromate workers with heart disease was 59.1 years, whereas that of the nine colored workers with heart disease was 43.3 years. The higher prevalence and

earlier appearance of syphilitic heart disease accounted for this difference. No correlation could be established between prevalence of heart disease and years in chromate industry. However, the mean vital capacity for chromate workers with heart disease is slightly lower than for the total chromate workers.

*Blood pressure.*—Although blood pressure readings were made after five to ten minute rest periods with the worker seated, basal conditions as presented by the New York Heart Association *(94)* were not approximated. All the workers came in from their respective jobs at various periods of their shift.

Table 36 shows the percentage of chromate workers with hypertension according to three different standards. Under conditions of this examination the New York Heart Association standards would classify half or more of the white workers 50 years of age or over as hypertensive. Even at younger ages a considerable percent of the workers would fall into the hypertensive group. It will be noted that classification according to the standards of Master and Dublin *(95)* shows a relatively low percentage with hyperten-

TABLE 36.—*Percentage of chromate workers with hypertension, according to standards used by New York Heart Association, Master and Dublin and in a study of ferrous foundries, according to age, by color.*

| Age group (years) | White | | | Colored | | |
|---|---|---|---|---|---|---|
| | New York Heart Association | Master and Dublin | Foundry levels | New York Heart Association | Master and Dublin | Foundry levels |
| All ages | 40.9 | 10.1 | 19.2 | 33.1 | 13.3 | 19.2 |
| Under 25 | 14.3 | 5.7 | 0 | 20.0 | 0 | 0 |
| 25–29 | 22.8 | 12.3 | 5.3 | 12.9 | 9.7 | 6.5 |
| 30–34 | 26.2 | 14.3 | 13.1 | 13.5 | 5.4 | 5.4 |
| 35–39 | 20.5 | 8.4 | 8.4 | 38.6 | 18.2 | 18.2 |
| 40–44 | 30.1 | 7.2 | 7.2 | 31.0 | 28.6 | 28.6 |
| 45–49 | 27.8 | 5.7 | 9.4 | 37.2 | 7.1 | 19.0 |
| 50–54 | 50.0 | 8.8 | 27.9 | 56.7 | 20.0 | 36.7 |
| 55–59 | 69.2 | 21.8 | 46.2 | 37.5 | 12.5 | 25.0 |
| 60–64 | 70.6 | 4.4 | 41.2 | 50.0 | 0 | 25.0 |
| 65 or over | 86.1 | 5.6 | 25.0 | 100.0 | 0 | 66.7 |

TABLE 37.—*Mean systolic and diastolic blood pressures of chromate workers, according to age, by color.*

| Age group (years) | White | | | Colored | | |
|---|---|---|---|---|---|---|
| | Number workers | Systolic (mm Hg) | Diastolic (mm Hg) | Number workers | Systolic (mm Hg) | Diastolic (mm Hg) |
| All ages | 646 | 135.3 | 85.4 | 240 | 131.5 | 85.1 |
| Under 25 | 35 | 121.8 | 78.1 | 5 | 122.4 | 76.4 |
| 25–29 | 57 | 126.2 | 80.7 | 31 | 121.4 | 79.4 |
| 30–34 | 84 | 126.5 | 83.0 | 37 | 121.8 | 78.6 |
| 35–39 | 83 | 126.9 | 84.7 | 44 | 131.8 | 87.0 |
| 40–44 | 83 | 130.0 | 85.0 | 28 | 133.6 | 89.6 |
| 45–49 | 54 | 129.3 | 85.5 | 42 | 132.4 | 83.9 |
| 50–54 | 68 | 140.1 | 89.1 | 30 | 141.9 | 92.7 |
| 55–59 | 78 | 149.3 | 91.2 | 16 | 142.8 | 88.5 |
| 60 or more | 104 | 152.2 | 86.4 | 7 | 146.9 | 87.7 |

sion at the older ages, but an increased prevalence among persons 25–34 years of age. The foundry study standards *(79)*, when applied to chromate workers, reveal large hypertensive groups at the older ages and at 30–34 years of age for white males and at 40–44 years for colored males.

Neither occupation in nor exposure to chromates appears to influence the prevalence of hypertension.

Table 37 shows the blood pressure readings for white and colored chromate workers.

## Abdomen

Fourteen chromate workers had enlarged livers on abdominal examination. These workers are older by 15 years than those without enlarged livers and have worked longer in chromates by an average of four years. However, neither age, nor time spent in chromate production correlates with degree of enlargement of the liver. The median age and time in chromates for workers with slight enlargement are higher (57.5 years and 26.3 years, respectively) than those for workers with livers enlarged to a greater degree (57.2 years and 9.4 years, respectively). The number in the entire group is too small to make any comparisons with alcohol consumption or environmental factors.

Effects on the liver have been described only in acute poisoning from potassium bichromate. Fatty degeneration of the liver is reported to have occurred in the fatally stricken cases in the Breslau episode. In a near-fatal case in this country, studied by Goldman and Karotkin *(53)*, it was noted that on the second day after ingestion of the poison the liver became tender, rigid, and enlarged. No jaundice developed, however, and the liver on examination four days later appeared to have returned to normal.

## Skin

During the conduct of the study the workmen were examined for dermatologic effects of chromic acid and its salts. The main types of dermatoses observed in this industry were: Chrome ulcers of the skin due to contact with chromic acid, sodium and potassium chromate and bichromate, and ammonium bichromate; and contact type dermatitis of the known ulcerative form due to the primary irritant, the sensitization effects of chromic acid and its salts, or both.

The frequent occurrence of chrome ulcers on the skin of workers exposed to chromic acid and its alkaline salts is well known. This type of lesion occurs more readily if there is a break in the continuity of the skin such as an abrasion, scratch or a laceration *(2, 6, 11, 97)*. The break in the skin becomes exposed to the chrome compound and the typical lesion ensues. The lesion has been described as being a round, nonspreading, deeply penetrating ulcer which has a hard, well-defined, circular and raised border. The central crater is clean cut and leads downward to a base covered with exudate or tenacious crust. Once a chrome sore has developed it is slow to

heal, and if exposure continues, it may persist for months. The healing process invariably leads to scar formation which is flat and atrophic. The lesion begins as a painless papule of pinhead size that gradually enlarges to form the mature lesion which may vary from 3 to 10 mm in diameter and may extend to considerable depth *(12)*.

There is no unanimity of opinion as to whether or not the ulcers are painful. It is conceded that the location of the ulcer over points of prominence such as the knuckles predisposes to a more painful process *(96)*.

In the six chromate-producing plants, workers were examined for dermatologic lesions with particular reference to ulcers and scars. It was noted that 300 or 45.9 percent of the white workers examined had ulcers or scars thereof compared with 151 or 61.9 percent of the colored workers. Considering white workers with these findings, 84.0 percent showed healed scars only, 5.0 percent had nearly-healed lesions, and 11.0 percent had active ulcerations. For the colored workers affected, the corresponding percentages were 82.1, 5.3, and 12.6, respectively. Thus, although colored workers had a higher prevalence of lesions, the distribution according to type was about the same as among white workers. The overwhelming majority of the active and the nearly-healed lesions had occurred within the six months previous to examination. Approximately 80 percent of all the lesions occurred on the hands, arms, legs, ankles and feet.

Among the entire 897 workmen examined only 17 presented any lesions which were suggestive of a chrome dermatitis. Most of these individuals presented an erythematous and papular type of dermatitis at points of contact, predominantly the upper and lower extremities.

It was noted that in most of the plants there existed adequate washing facilities, and facilities for providing clean working clothes to workers. These preventive measures probably accounted for the low prevalence of dermatologic lesions and, especially, the absence of the chronic lesions described in earlier reports.

## Blood

The possible effects of chromium compounds on the blood have not been extensively studied. Blood changes as recorded by Brieger and others in the Breslau acute poisonings *(55, 64)* consisted essentially of evidence of bone marrow stimulation with intense leukocytosis—in one case reaching 41,900 leukocytes per cubic millimeter of blood—with immature polymorphonuclear cells, myelocytes, and myeloblasts. Nucleated red blood cells and Howell-Jolly bodies were seen. The blood platelets were increased to as high as 421,000 per cubic millimeter of blood, but no clotting change was noted.

On the other hand, studies of those chronically exposed have not yielded results nearly as striking. Lieberman *(38)* in a study of five chromium platers reports no blood abnormalities, although the specific tests performed were not indicated. Mancuso *(34)* found a slightly increased leukocyte

Figure 8.—Perforation of nasal septum revealing zones of necrosis and marked irritation of adjoining hyperemic mucosa.

Figure 10.—Discoloration of teeth in a chromate worker.

Figure 9.—Perforation of nasal septum, medium size, anterior margin

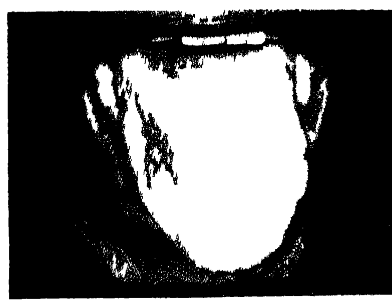

Figure 11.—Discoloration of tongue of a chromate worker

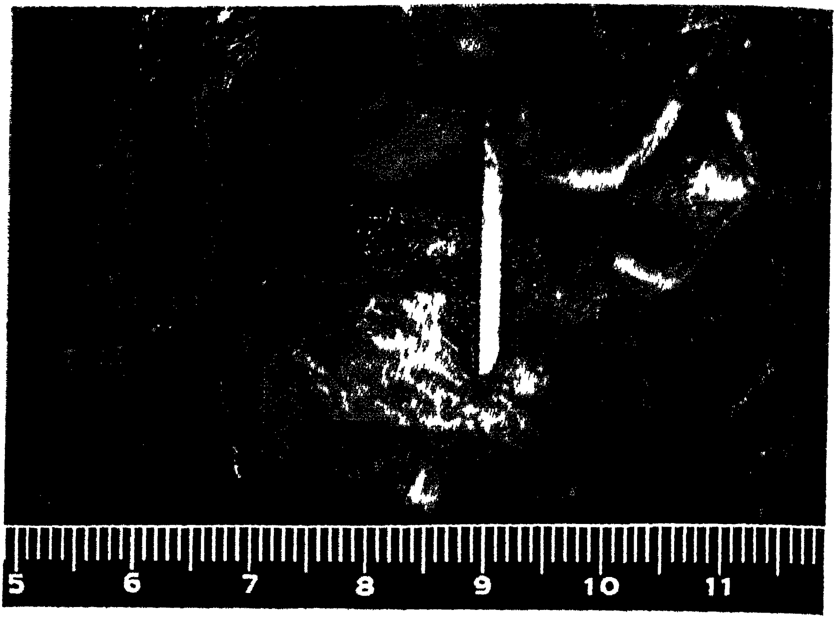


Figure 12.—Case Number 1. Anaplastic squamous cell carcinoma of anterior segmental bronchus of left upper lobe

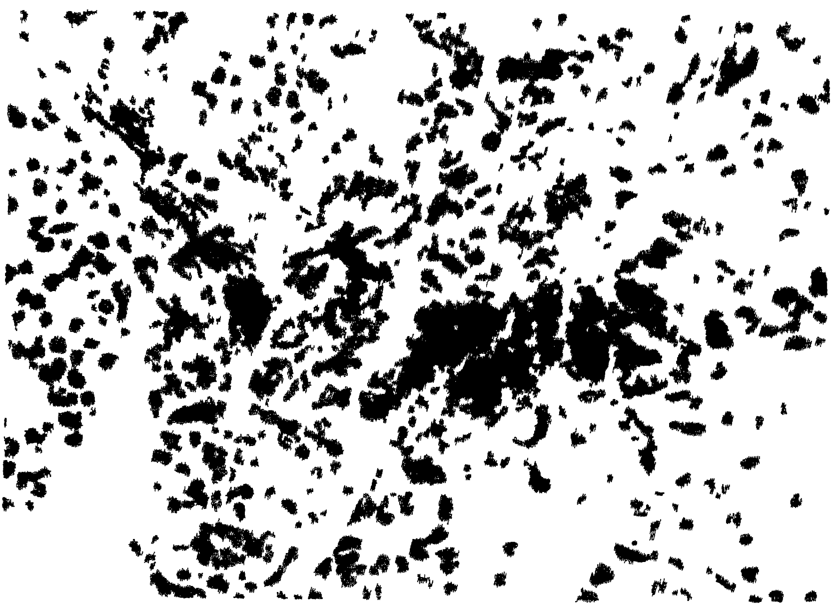

Figure 13.—Case Number 1. Lung specimen, large deposits of brownish black amorphous particles among anaplastic cells

Figure 14.—Case No 1

Figure 15 —Case No 2

**Figure 16.—Case No. 2**

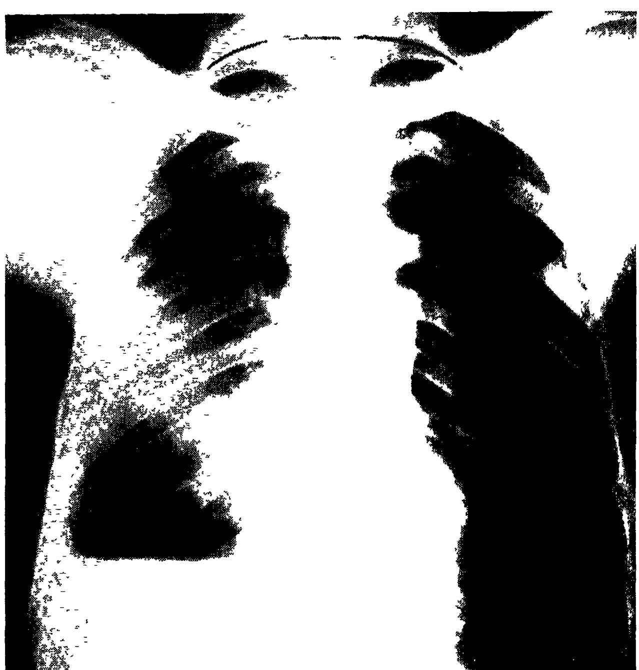

**Figure 17.—Case No. 2**

**Figure 18.—Case No. 3**

**Figure 19.—Case No. 4**

Figure 22.—Case No. 7

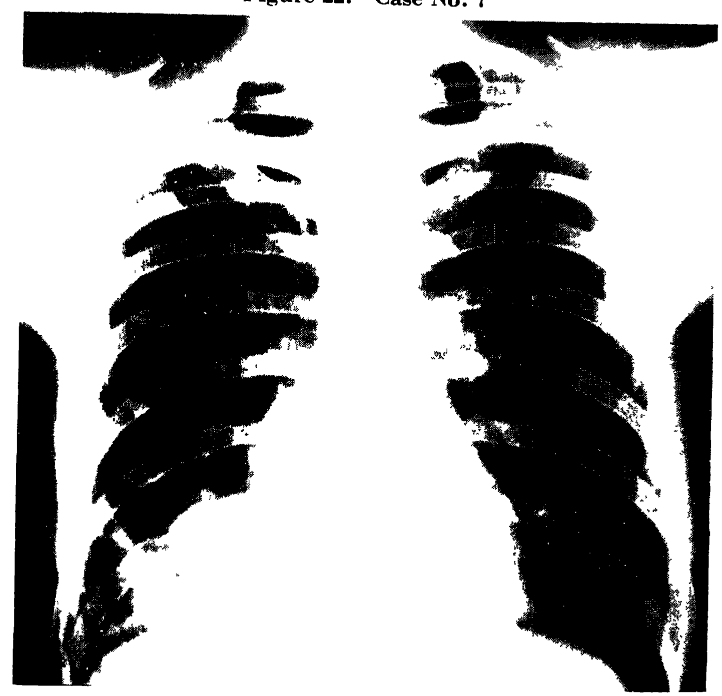

Figure 23.—Case No. 8

**Figure 24.—Case No. 9**

**Figure 25.—Case No. 10**

count, a questionable increase in monocytes and eosinophils, a slightly increased bleeding time, and possibly a slightly decreased hemoglobin level. The red blood cell count, coagulation time and blood protein values were all essentially normal.

Some abnormalities in the blood have been detected in experimental chromium poisoning. Thus, Priestley *(63)*, in 1877, noted that in guinea pigs given lethal doses of potassium chromate, the blood gave an absorption spectrum like that of methemoglobin, and Mattuci *(65)* reproduced this phenomenon in vitro, using potassium bichromate. Mauro *(98)*, on the basis of his work on rabbits with chromic oxide, believed that exposure to these substances in large amounts produced anemia, due to the inhibition of erythropoietic function.

### *Red blood count*

The median erythrocyte count was 4,930,000 for all workers, slightly higher in the colored group than in the white. There was a slight decrease in red blood cell count with years worked in chromate. These results are comparable with the findings in the ferrous foundrymen *(79)* and open-hearth steelmen *(80)*.

### *Hemoglobin*

The median hemoglobin estimations for the white chromate workers is 15.7 gm percent while that of the colored chromate workers is 16.3 gm percent. No change is observed with exposure to chromates.

### *Hematocrit*

The hematocrit median value is 47.1 for white workers, as compared with 45.8 for colored chromate workers. Lower readings were found in those workers with longer time in chromate; the colored chromate workers showed this effect on the average ten years earlier than did the white chromate workers. No significant differences are manifest in hematocrit levels between foundrymen and chromate workers.

### *Sedimentation rate*

The corrected sedimentation rates for blood of chromate workers by color and years in chromate industry are shown in Table 38. The median rate increases with years in the chromate industry from 9.0 mm for white workers with less than 10 years' experience to 14.0 mm when these workers had been 20 years or more in this industry. The medians for the colored workers increased from 11.7 mm to 15.0 mm during this period. These values are higher than those of foundrymen *(79)*. When the two colors are combined the median sedimentation rate for all workers is 11.3 mm; for those working less than 10 years it is 10.0 mm, for those working 10–19 years it is 12.6 mm and for workers with 20 years or more in this industry it is 14.3 mm.

### *White blood count*

The total leukocyte count reveals a median of 8,824 for all the chromate

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**Figure 20.—Case No. 5**

**Figure 21.—Case No. 6**

Figure 22.—Case No. 7

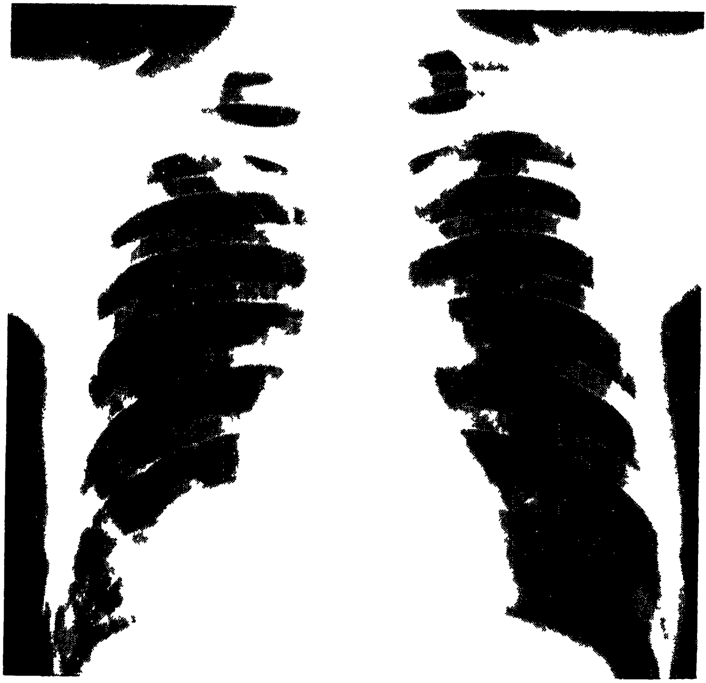

Figure 23.—Case No. 8

Figure 24.—Case No. 9

Figure 25.—Case No. 10

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TABLE 38.—*Corrected sedimentation rates of erythrocytes for chromate workers, by time in chromate industry and color.*

| Corrected sedimentation rate (mm in 1 hour) | Total | Time in chromate industry: Less than 10 years | 10–19 years | 20 years or over |
|---|---|---|---|---|
| | White | | | |
| Total | 644 | 350 | 155 | 139 |
| Under 10 | 299 | 194 | 61 | 44 |
| 10–19 | 250 | 120 | 67 | 63 |
| 20 or over | 95 | 36 | 27 | 32 |
| Median | 10.9 | 9.0 | 12.5 | 14.0 |
| | Colored | | | |
| Total | 242 | 171 | 47 | 24 |
| Under 10 | 102 | 75 | 18 | 9 |
| 10–19 | 86 | 61 | 19 | 6 |
| 20 or over | 54 | 35 | 10 | 9 |
| Median | 12.2 | 11.7 | 12.9 | 15.0 |

workers. The difference in count for white and colored chromate workers was not appreciable and comparison with foundry workers revealed no significant difference.

### *Serological test for syphilis*

Blood specimens were submitted to local health departments for serological test for syphilis. The tests used (Eagle-Strauss, Mazzini and New York State) varied according to the respective health departments. Among the white chromate workers 1.6 percent showed a positive serological test for syphilis whereas the colored chromate workers showed 14.8 percent. These percentages rise to 1.9 and 20.7 when "doubtful" cases are added. The nonchromate workers yielded 1.4 percent positive among white, and 28.6 percent among colored workers. Other surveys reveal percentages of the same general magnitude *(99)*; foundry workers *(79)*, 2.1 percent white and 10.3 percent colored; and open-hearth steel workers *(80)*, 1.6 percent white and 23.8 percent colored.

## Urine

### *Sugar and albumin*

Examination of the urine for sugar and albumin revealed about the same prevalence as found in other surveys. However, white and red blood cells and casts appeared with greater frequency than is usually observed in the average industrial population.

### *Sediment*

The frequency of white blood cells in urine of chromate workers, according to the years of exposure in chromate, shows an increase with years for both colors as revealed by a higher percentage of workers with over five cells per high-power field.

Casts in urine were found in a greater percentage of workers who have worked 10 years or over than in those working less than 10 years. Red blood cells were found in 2.3 percent of white and 6.5 percent of colored workers. These percentages did not change appreciably with years in chromate exposure.

## Miscellaneous laboratory procedures

### *Chromium*

Urine and blood samples were analyzed for chromium content. The median value for chromium in urine as determined from 222 samples was 0.043 mg Cr per liter for white males and 0.071 mg Cr per liter for colored males. Values less than 0.010 were found in 5.0 percent of all of the samples and values of 0.150 or over were found in 15.3 percent. No consistent differences were noted with respect to job exposures.

A total of 77 samples was available for the determination of chromium in the blood. The median value for blood of white males was 0.004 mg percent and of colored males it was 0.006 mg percent. Seventy-nine percent of all samples fell between 0.002 and 0.010 mg percent. Twelve percent of the samples had values of 0.010 or over.

Blood chromium levels in Alwens' cases *(70)* were high in two individuals with cancer, whereas a chromate worker who did not have cancer was found to have only a trace of chromium in his blood. Letterer and his associates *(44)*, on the basis of their studies on a small series of cases, felt that there was no relationship between the amount of chromium in the tissues and the duration of exposure, or the presence or absence of cancer.

### *Coproporphyrins*

The finding of coproporphyrinuria has been observed in poisoning of such industrial hazards as lead, aniline and arsenic as well as in toxicity due to certain drugs such as acetophenetidin, acetanilid, acetyl-salicylic acid, sulfonamide compounds, ether, and paraldehyde and morphine. This finding has also been noted in such pathological states as cirrhosis of liver, familial hemolytic jaundice, pernicious anemia and pulmonary infections with suppuration and high fever *(100, 101)*. All urine samples from chromate workers were found to be within the normal limits of 100–120 micrograms per 24 hours.

### *Mucoproteins and polysaccharides*

An increased serum mucoprotein content has been observed in a variety of infectious and neoplastic diseases *(102)*. In neoplasia this increased mucoprotein level manifests a relatively normal ratio of polysaccharides to proteins (Pm/M).

Sera of 62 chromate workers were submitted for study. The determinations were made by the method of Winzler and associates *(103, 104)*. Of the 62 cases, 20 chromate workers had mucoprotein levels above 75.4

mg/100 milliliters serum and 5 had levels below 35.6 mg/100 milliliters serum, both being outside the normal range for males, which is 48 to 70 mg/100 milliliters. Sixteen chromate workers had polysaccharide levels above 140 mg/100 milliliters serum which is considered to be above normal. It is interesting to note that all three chromate workers who were in this series, and who were known to have bronchiogenic carcinoma, had high levels for mucoproteins and polysaccharides. Further work is necessary to evaluate the significance of these determinations as a screening aid for pulmonary malignancy.

## Pulmonary neoplasms

### *Findings*

Of the 897 chromate workers examined ten have been considered as having bronchiogenic carcinoma. Of these, eight have in addition to roentgenological and clinical findings, histological evidence as obtained by pneumonectomy in 5 cases, biopsy in 1 case, necropsy in 1 case, and bronchial washings Class V in 1 case. Of the remaining two, one has died. He was diagnosed one month before death in a hospital as having roentgenological and clinical findings of bronchiogenic carcinoma. The other was admitted to a hospital with the chief complaint of weakness; extension of lesion on chest film was found, as well as a red blood cell count of 1,700,000.

These ten cases can be classified as follows:

### *(1) Proved cases*

In addition to roentgenological findings, pathological material confirmed the diagnosis.

*Early.*—Not diagnosed at the time of survey.

Case No. 1, pneumonectomy, anaplastic squamous cell carcinoma.

Case No. 2, pneumonectomy, squamous cell carcinoma.

Case No. 3, pneumonectomy, squamous cell carcinoma.

Case No. 4, pneumonectomy, squamous cell carcinoma; died June, 1951.

Case No. 5, necropsy, squamous cell carcinoma; died February, 1952.

*Late.*—Diagnosed as bronchiogenic carcinoma before time of survey.

Case No. 6, bronchial washings, Class V.

Case No. 7, thoracotomy, biopsy squamous cell carcinoma.

Case No. 8, pneumonectomy, squamous cell carcinoma.

### *(2) Radiological evidence only*

*Early.*—Not diagnosed at time of survey.

Case No. 9, died July 1951, no necropsy.

Case No. 10, hospitalized, under observation, bronchiogenic carcinoma.

Table 39 summarizes the pertinent information on these 10 cases. The mean age is 54.5 years and the mean exposure to chromates is 22.8 years. Three cases did not have perforation of the nasal septum. Sedimentation rate was increased in all cases, with a mean of 25.2. Of the eight cases with X-ray films none showed lung markings beyond linear two.

In addition Case No. 11 (not in the survey) is described because it

TABLE 39.—*Pertinent information on 10 cases of cancer of the lung among chromate workers.*

| Case number | Age (years) | Years in chromate industry | Color | Perforation of nasal septum | Occupation at time of survey | Dyspnea | Chest pain | Cough[1] | Chest X-ray | Red blood count (millions per mm $^3$) | Total leucocyte count per mm $^3$ | Corrected sedimentation rate (mm in 1 hr.) | Albumin in urine (mg) | Casts in urine |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | 43 | 21 | C | Yes | Soda operator | No | No | No | L1 | 3.6 | 7,900 | 22 | Positive 5 | 1–2 coarse granular. |
| 2 | 45 | 18 | C | Yes | C A cooker | No | No | No | L2 | 4.1 | 15.600 | 21 | do | No. |
| 3 | 62 | 39 | W | Yes | Kiln burner | No | No | No | L1 | 5.3 | 14,000 | 22 | Negative | No. |
| 4 | 62 | 25 | W | No | Laborer | Moderate. | Yes | Productive cough | L2 | 3.3 | 21,900 | 28 | do | 0–2 waxy. |
| 5 | 53 | 14 | W | No | Training supervisor. | No | Yes | do | Normal | 5.3 | 11,200 | 25 | do | 0–2 fatty. |
| 6 | 63 | 22 | W | Yes | Cooper | No | No | No | No film | 3.7 | 15,600 | 33 | Positive 10 | 0–1 coarse granular. |
| 7 | 62 | 27 | W | Yes | Gateman | Mild | No | Productive cough. | L2 | 4.8 | 10,600 | 22 | Negative | No. |
| 8 | 53 | 21 | W | No | Foreman | Mild | No | do | No film | 4.8 | 7,500 | 16 | do | No. |
| 9 | 48 | 8 | C | Yes | Kiln room | No | No | No | Normal | 5.2 | 4,100 | 35 | do | Not stated. |
| 10 | 54 | 33 | W | Yes | Laborer | No | No | Productive cough. | L1 | Not done | | 28 | do | No. |

[1] None had hoarseness.

presents a diagnostic problem. Roentgenological evidence and biopsy reports are presented.

### *Pathological considerations*

The materials which have been considered by different investigators to be responsible for the increased incidence of pulmonary cancers in chromate workers have been the bichromates, the monochromates, free chromic acid and the chromites.

Gross *(72)*, as well as Machle and Gregorius *(35)*, considered the monochromates to be the compounds responsible for lung cancer. Bauer *(105)* thought the responsible substance was the free chromic acid or the alkali salts, especially the bichromates. Koelsch *(76)* believed that the monochromates and bichromates were the responsible factors in pulmonary cancers, and that the chromites were not harmful since they are scarcely soluble in the body. Gross and Koelsch *(73)* believed that zinc chromate among pigment workers was the cause of lung cancer because it was more soluble than barium or lead chromate.

However, Mancuso and Hueper *(78)* gave serious consideration to chromite ore as a potential carcinogenic agent, and further added to this consideration chromium pigments and chromium alloys. They believe that the insoluble chromium compounds are retained in the lung over long periods of time and may give rise there to the production of pneumoconiotic changes.

The presence of "anthracotic particles" in the lung of chromate workers has been frequently pointed out. However, the presence or insolubility of a substance in the lung does not incriminate that substance as a carcinogenic agent. It should be observed that silica dust, which produces marked irritation and stimulates the growth of fibrous tissue, does not predispose to cancer of the lungs *(106)*.

In order to further evaluate the role of chromite ore in the production of pulmonary cancer, a refractory plant using chromite ore to make chromite brick was investigated. An engineering survey evaluated the exposure to chromite ore in the refractory plant and found it to be equivalent to that which occurs in the chromate-producing industry. An analysis of the death certificates of all employees of that plant was made. A record of the deaths of all employees from all causes at the chromite refractory plant during the 14-year period, 1937–1950, gives a death rate of 635.2 per 100,000 males aged 15–74 years as shown in Table 40. On the basis of the chromate-producing company death rate instead of the 43 deaths actually found, the total would have been 102 deaths. Had the rate for all United States males applied there would have been 63 deaths.

Among the refractory workers there were the following cancer deaths: one cancer of testes, one stomach cancer and one lung cancer. Instead of 3 cancer deaths there would have been 9 deaths if the United States rate had been followed. Had the chromate rate applied deaths would have numbered

TABLE 40.—*Death rate and number of deaths according to cause among male workers in a chromite refractory plant compared with the expected number based on the death rate for chromate plants and for the United States.*

| Cause of death | Chromite refractory plant | Chromate plants | United States |
|---|---|---|---|
| | Rate per 100,000 population | | |
| All causes | 635.2 | 1,503.1 | 927.8 |
| Cancer of respiratory system | 14.8 | 470.8 | 16.7 |
| Cancer other sites | 29.5 | 108.7 | 113.6 |
| Diseases of the heart | 73.9 | 398.4 | 355.3 |
| All other diseases [1] | 517.0 | 525.2 | 442.2 |
| | Number of deaths | | |
| | Actual | Expected [2] | Expected [3] |
| All causes | 43 | 101.8 | 62.8 |
| Cancer of respiratory system | 1 | 31.9 | 1.1 |
| Cancer other sites | 2 | 7.4 | 7.7 |
| Diseases of the heart | 5 | 27.0 | 24.1 |
| All other diseases [1] | 35 | 35.5 | 29.9 |

[1] Including unknown causes.
[2] Based on rate prevailing in chromate plants.
[3] Based on rate prevailing in United States males 15–74 years of age, 1940–48.

39. Cancer of the lung appeared once, or exactly the same as would be expected from applying the United States rate. Had the chromate rate been followed there would have been 32 deaths from lung cancer. Because of a lack of pertinent information, no attempt is made to explain the striking data yielded by diseases of the heart.

It is to be pointed out that at the pH of the body, the bichromates would be rapidly converted to monochromates, also, exposures at different work stations of the plants reveal varying mixtures of the monochromates, bichromates as well as chromites in nearly all instances. In addition, many workers have rotated jobs, and other jobs entail different degrees of exposure at various phases of the work. In most instances past records of environmental studies were lacking. During the past years the industry has made numerous improvements in equipment which have undoubtedly reduced the exposure. Hence, correlation of environmental studies with medical findings has been difficult in this respect.

In several reports *(34, 76, 78)* reference has been made to the relationship of perforation of the nasal septum and pulmonary cancer. The present study has shown that prophylaxis carried out by the individual worker can markedly affect the rate and time of onset of septal perforations due to chromates. This study has further shown that the chromate workers have a prevalence of 56.7 percent for perforations of the nasal septum. This high prevalence makes correlation difficult with the lower prevalence for pulmonary cancer. There have been several cases with pulmonary carcinoma in chromate workers who did not have nasal perforations. Neoplasms arising from the site of the nasal perforation or chromium dermatitis have not been reported. Thus, the use of the prevalence of nasal perforations as an index of the prevalence of pulmonary carcinoma is not valid.

Experimental work has been carried out by several investigators to study this problem in greater detail. Lukanin *(25)* placed rabbits and cats in various areas of a chromate plant for 2–8 months. One of the animals developed nasal septum perforation but no mention was made of any pulmonary neoplasm. Lehmann *(66)* exposed animals to a spray of bichromate and was able to produce a septal perforation but no mention is made of any pulmonary neoplasm. Shimkin *(107)* injected chromite ore intravenously into cancer-susceptible mice, and found no increase as compared with controls. In Alwens' report *(70)* mention is made that Gross was unable to produce bronchial carcinoma in animals through the inhalation of chrome dust over long periods, although one animal showed hyperplasia of the hilar glands. Akatsuka and Fairhall *(41)* did not report any neoplasms after the ingestion and inhalation of chromium carbonate and chromic phosphate for 1–3 months.

However, Schinz *(108)* was able to produce sarcomas in the thighs of rabbits four years after implants of metallic chromium.

Alwens *(70)* reported that 79 percent of his cases were squamous epithelial carcinomas and 21 percent were adenocarcinomas. Hueper *(109)* reviewed the German cases as 4 squamous cell, 2 round cell, 2 adenocarcinoma and one oat cell carcinoma. Baetjer *(110)* reports that in seventeen cases six were oat cell, five squamous, 4 undifferentiated and two anaplastic type of carcinoma.

### *Chemical analyses for chromium*

Alwens *(70)* and Mancuso and Hueper *(78)* have determined the amount of chromium in the lung of chromate workers.

The following is a chemical analysis of a lobe of the lung in case No. 1:

| Item | "Normal" tissue | | Carcinoma tissue | | Notes |
|---|---|---|---|---|---|
| Tissue: | | | | | |
| Weight of tissue received | 185.0 | grams | 0.62 | grams | 1 |
| Weight taken for analysis | 10.9 | grams | 0.62 | grams | 1 |
| Dry residue (108°C.) | 17.5 | percent | – | | 2 |
| Ash residue (550°C.) | 0.8 | percent | 0.9 | percent | – |
| Acid insoluble ash | 0.09 | percent | 0.10 | percent | 3 |
| Acid insoluble chromium | 20.0 | gamma/g | 11.0 | gamma/g | 4 |
| Acid soluble chromium | 3.3 | gamma/g | 4.0 | gamma/g | 4 |
| Sodium | 0.46 | mg/g | 0.81 | mg/g | 5 |
| Potassium | 0.31 | mg/g | 0.68 | mg/g | 5 |
| Preservative media: | | | | | |
| Acid soluble chromium | 0.019 | gamma/ml | 0.014 | gamma/ml | 6 |
| Sodium | 0.40 | mg/ml | 0.37 | mg/ml | 6 |
| Potassium | 0.28 | mg/ml | 0.29 | mg/ml | 6 |
| Ml preservative per gram of tissue | 3.19 | | 71.6 | | – |

NOTE 1.—These weights are the weight of tissue after squeezing and blotting with blotting paper to remove as much of the moisture as possible by this method.

NOTE 2.—The *normal* lung tissue was dried overnight at 108°C.

NOTE 3.—After ashing at 550° C. the ash was treated with dilute nitric acid. Material which was not dissolved was considered as acid insoluble. This portion was analyzed for acid insoluble chromium.

NOTE 4.—These concentrations are expressed as gammas (micrograms) of chromium per gram of tissue on the basis of the blotted weight.

NOTE 5.—Sodium and potassium in the tissue are expressed as milligrams of the elements per gram of blotted tissue.

NOTE 6.—The formalin preservative solution was analyzed for chromium, sodium and potassium. These concentrations are expressed as gammas (micrograms) or milligrams per milliliter of the solution.

The following is a chemical analysis for chromium (as Cr) of tissues obtained from the necropsy of case No. 5:

| Tissue | Chromium (mg/100 g. of blotted wet tissue) |
|---|---|
| Brain | 0.005 |
| Cancerous tissue | .000 |
| Kidney | .003 |
| Liver | .023 |
| Spleen | .012 |
| Skeletal muscle | .004 |
| Left lung | 1.2 |
| Bone (spinal) | .046 |
| Blood | .001 |
| Pericardial fluid | .000 |

NOTE 1.—In connection with the value (1.2) obtained for the lung it is noted that because of possibility of chromite ore entering the lung, a second portion of lung tissue was ashed and a fusion made to dissolve chromite ore. By this method a value of 5.8 mg of chromium per 100 grams of tissue was obtained, indicating the presence of considerable chromite ore.

NOTE 2.—It is noteworthy that chromium concentration is relatively high in both bone and liver, which suggests some form of storage in these two tissues and suggests further a metabolic characteristic of chromium in the body.

### Case histories — Proved cases
### *(not diagnosed as cancer at time of survey)*

*Case No. 1.*—Colored male, 43 years old. See Figures 12, 13, and 14.

*Employment History:*

*Chromate.*—From 1933 to present date patient was a granulator soda operator. From 1931 to 1933 patient worked in the shipping department, handling drums of bichromate and chromates.

From 1929 to 1931 patient worked as lime mill operator.

*Nonchromate.*—From 1921 to 1929 patient was a truck driver for a canning factory.

*Medical History:*

*Habits.*—Smoked an average of 10 cigarettes a day.

*Previous Illness.*—The patient developed a perforated nasal septum after 2 years of employment in chromate industry. In 1940, he had three chrome ulcers on right hand. The patient had pneumonia in 1943.

*Previous Medical Examinations.*—Preplacement and periodic examinations during the past 15 years were not significant in their findings. Chest film revealed accentuation of hilar structures and some increase in bronchovesicular structures and was read as normal.

On August 2, 1950, the Public Health Service survey team examined this patient and some of the pertinent findings were: nasal perforation with crusting; no chest pain, hoarseness, cough or hemoptysis; weight 157 lbs., weight a year ago 155 lbs.; congestion of nasal turbinates and conjunctiva; redness of throat.

*Blood.*—Erythrocyte count 3,670,000; hemoglobin estimation 12.5 grams percent; hematocrit 44 percent; corrected sedimentation rate 22 millimeters at the end of one hour; white blood cell count 7,900.

*Urine.*—Albumin 5 milligrams; white blood cells per high-power field 35-50; 1-2 coarsely granular casts.

*Chest film.*—Suspicious hilar markings on left. Follow-up needed.

*Present Illness.*—At the end of November and the beginning of December 1950 the patient had a mild chest cold associated with a chronic cough. This gradually got better until the latter part of February 1951 when the patient had an attack of pneumonia which cleared up with treatment by the latter

part of March 1951, but the cough persisted in a somewhat diminished degree. The patient lost a few pounds of weight during the attack of pneumonia but gained it back shortly thereafter. Hemoptysis, night sweats, anorexia and increase in fatigability were not present.

The patient was away from work on March 30, 1951, to April 10, 1951, with a diagnosis of "grippe and bronchitis" by his private physician. Upon his return to work he was X-rayed; a suspicious shadow was revealed in left hilar area. This shadow was interpreted at the time as more typical of pneumonia or pneumonitis. On April 11, 1951, he was examined by an internist whose significant findings were: chronic cough, pain in shoulder, temperature 100.4. Examination of lungs revealed bronchial type of breathing in left hilar region. Sedimentation rate 8 mm. Fluoroscopy revealed enlarged hilar shadow and a "nest" over left hilus. Patient should be bronchoscoped. Pain in the shoulder was interpreted to be due to arthritic changes in cervical vertebrae. On April 24, 1951, film series were reviewed by consultant radiologist who diagnosed the patient as having bronchiogenic carcinoma. On April 25, 1951, he was bronchoscoped with the finding of no evidence of growth, and the diagnosis of "bronchitis, not too severe" was made. The bronchial washings were read as normal, Class I.

Chest X-rayed monthly. Oblique film (June 5, 1951, fig. 14) reveals lesion to be rather well defined and demarcated, about the size of an English walnut and showing some reactivity about its periphery. It lies just in front of and above the left hilum, and related to major bronchus of left upper lobe.

On July 6, 1951, he was bronchoscoped again and was found to have distortion with fixation of the left main stem bronchus. Pathologic study of secretions and biopsy were negative. Fluoroscopy on July 7, 1951, revealed obstruction of the aortic window which failed to open with deep inspiration. On July 9, 1951, a tuberculin test (1–1,000) was negative.

On the afternoon of July 11, 1951, patient experienced a violent coughing episode with generalized pain in left chest. A physical examination shortly afterwards revealed generalized suppression of breath sounds with a tympanic note on percussion throughout the left chest and a pneumothorax was suspected. This was later confirmed by X-ray. Fluoroscopy on July 12, 1951, revealed a "mantle type" pneumothorax on left, but lesion was of the same size and location.

On July 17, 1951, patient was admitted to hospital with complaint of constant burning sensation of two weeks' duration substernally and extending to the left second interspace. Physical examination revealed suppression of breath sounds over the left upper chest anteriorly and prolongation of the expiratory phase and persistent expiratory rales near the hilar area. On July 17, 1951, an exploratory thoracotomy was performed. The left lung was removed at which time a bronchiogenic carcinoma was found in the anterior segment of the left upper lobe. The postoperative course was uneventful and he was discharged on July 25, 1951.

The final diagnosis is anaplastic squamous cell carcinoma of the round to oat cell type, primary within the anterior segmental bronchus of the left upper lobe, with metastasis to a left hilar lymph node. The final disposition was thus a palliative pneumonectomy with guarded prognosis.

The chemical analysis for chromium in the removed lung appears under **Pulmonary neoplasms**, page 92.

*Case No. 2.*—Colored male, 45 years old. See Figures 15, 16, and 17.

*Employment History:*

*Chromate.*—Since 1945 patient has been a cooker and helper in the chromic acid department.

He was a liquor boiler operator (sodium dichromate liquor) from 1939 to 1945. From 1934–39 he was a packer of crystalline soda bichromate. From 1932–34 he was a washer of crystalline soda bichromate.

*Nonchromate.*—From 1931 to 1932 patient was a laborer loading scrap iron into cars. From 1925–31 he worked as chauffeur for a meat company. From 1920–25 he was a laborer (section hand) on a railroad.

*Medical History:*

*Habits.*—Smoked an average of 10 cigarettes, 5 pipefuls; had one chew per day.

*Previous Illness.*—Patient developed a perforated nasal septum after one year of exposure to chromates. Only other illness, "La grippe" on March 24, 1940, November 4, 1944, November 3, 1946, and on February 7, 1950.

*Previous Medical Examinations.*—His preplacement and past periodic examinations did not reveal any abnormalities, except for the presence of a perforated nasal septum.

On March 24, 1948, patient had his chest X-rayed in the plant, for the first time, which was read as negative. On September 24, 1948 the patient had a semi-annual periodic examination with negative findings (fig. 15). On March 31, 1949, repeat semi-annual periodic examination did not reveal any abnormalities. On August 30, 1949, semi-annual periodic examination was described as negative.

On February 7, 1950, routine semi-annual examination was recorded as negative for both physical findings and chest film (fig. 16).

*Present Illness.*—On June 30, 1950, patient came into plant dispensary complaining of pain in right side of lower chest and was sent home. On July 1, 1950, he entered a hospital where he was diagnosed as having lobar pneumonia, placed on penicillin therapy, and was discharged on July 15, 1950, as cured. He convalesced for two weeks at home and then returned to work on August 4, 1950. His chest was X-rayed by the plant medical department on August 11, 1950.

On August 16, 1950, the patient was examined by the Public Health Service survey team with the following significant findings: no cough, hemoptysis, streaking, chest pain, or any other symptom referable to respiratory tract; epistaxis, streaking and nasal septum perforation with crusting and congestion of turbinates; healed chrome scars (active 10 years ago) on both hands, arms, forehead and right side of face; blood pressure 122/74; pulse 102 per minute; respirations 28 per minute; temperature 99.4° F.; height 71¾ inches; weight 156 lbs., weight 1 year ago 171½ lbs.

*Blood.*—Erythrocyte count 4,100,000; hemoglobin estimation 14 grams percent; hematocrit 37 percent; corrected sedimentation rate 21 millimeters at the end of one hour; white blood cell count 15,600.

*Urine.*—Albumin 5 milligrams per 100 milliliters of urine.

*Chest film.*—A wedge shaped density observed in right middle lobe region; mediastinum appears to be deviated to right. Further workup advised to rule out malignancy (fig. 17). Consultant group of roentgenologists and chest physicians for the Public Health Service reviewed the films (3 of which are reproduced here) as follows: One consultant felt that starting with film of March 24, 1948, an area of increased pulmonary markings is noted just a little above the cardiophrenic angle. There is increased density and widening as the films progress. Another consultant felt that

commencing with film No. 4, there are abnormal shadows in the fourth interspace extending down from the level of the 4th rib laterally to the right border of the heart through the fifth rib with some small clear areas between. All consultants agreed to the finding of the mass on film No. 5 (fig. 16), although the area has no dense homogeneity. On film No. 6, the area has density and extends from the second rib through the lower level of the fifth rib. In addition a pleural effusion has developed at the third interspace just above the fourth rib.

*Further Findings.*—On August 21, 1950, the patient was sent by plant physician to an internist who noted: 1. Perforated nasal septum. 2. Chest—lower right posterior area where relative dullness and some change in breath sounds and spoken voice are noticed. Fluoroscopy reveals a shadow in the right hilar region that should be watched. 3. Blood findings: Erythrocyte count 3,910,-000, white blood cell count 6,250, and 11 percent eosinophils.

Four days later on August 25, 1950, the patient was bronchoscoped, at which time a lesion was noted on the right side which was highly suggestive grossly of a tumor mass. Bronchial washings revealed "fairly good evidence of a malignant neoplasm Class IV."

On September 20, 1950, right pneumonectomy was done. It is to be noted that at this time the only complaint the patient offered was pain on the right side of chest, which he had experienced since he was bronchoscoped. He had no cough, nor hemoptysis. The findings were that of a bronchial carcinoma of the right lung, epidermoid, anaplastic, primary of the hyparterial bronchus, at its point of origin from the right main bronchus, with atelectasis of the right middle lobe, hemorrhage and pleural thickening of right lower lobe. The patient was discharged on October 6, 1950, apparently well with no symptoms. A checkup three weeks later revealed no new findings. At present the patient is a bus driver, apparently well.

*Case No. 3.*—White male, 62 years old. See Figure 18.

*Employment History:*

*Chromate.*—From 1927 to the present patient has worked as a kiln burner. He traveled during 1926–27. From 1912 to 1926 he was a hand furnace fireman. From 1910 to 1912 he worked as a maintenance man.

*Nonchromate.*—Prior to 1910 patient was a farmer.

*Medical History:*

*Habits.*—Did not smoke.

*Previous Illness.*—After one year of exposure to chromates, patient developed a perforation of the nasal septum. The patient has had numerous chrome ulcers which healed with very little scarring. Has an average of 2 colds a year.

*Previous Medical Examinations.*—Periodic examinations at the chromate plant were essentially negative, other than that of perforated nasal septum.

On July 24, 1950, the Public Health Service survey team examined the patient and found the following significant findings: no cough, chest pain or hemoptysis. Crusting, whistling, epistaxis and streaking of nose as well as perforation of the nasal septum; blood pressure 180/98; weight 175 lbs., weight 1 year ago 176 lbs.; old burn scars of belt line, feet, and hands.

*Blood.*—Erythrocyte count 5,310,000; hemoglobin estimation 17.5 grams percent; hematocrit 47 percent; corrected sedimentation rate 22 millimeters at the end of one hour; white blood cell count 14,000.

*Urine.*—White blood cells per high-power field 10–15.

*Chest film.*—Suspicious area of density in lower right lobe. Follow-up indicated.

*Present Illness.*—The patient has been asymptomatic although films have been

progressively more suggestive; e.g., fig. 18, Aug. 24, 1951. In September 1951 bronchoscopy was negative. However, bronchial washings were classified as V—Malignancy. On September 14, 1951, he had a pneumonectomy with finding of bronchiogenic carcinoma of right lung. A papillary tumor, the size of a "lima bean" was found in the bronchus to the middle lobe, occluding the middle section.

NOTE.—This patient had a high polysaccharide level in serum on June 15, 1951.

*Case No. 4.*—White male, 62 years old. See Figure 19.

*Employment History:*

*Chromate.*—From 1936 to the present time patient was a laborer. From 1933 to 1936 he was unemployed. From 1922 to 1933 he was a laborer.

*Nonchromate.*—From 1918 to 1922 he worked at furnaces. From 1912 to 1918 patient laid sewer pipes. From 1907 to 1912 he worked in a foundry as furnaceman. Prior to 1907 patient was a farmer

*Medical History:*

*Habits.*—Smoked 20 cigarettes a day.

*Previous Illness.*—Pneumonia in 1937

*Previous Medical Examinations.*—Periodic examinations at plant have not revealed any significant abnormalities.

*Present Illness.*—For the past 2 months patient has had "a cold" in chest with much productive coughing at night.

On November 28, 1950, patient was examined by Public Health Service survey team which revealed the following: impaired hearing for past three years; dyspnea for past two months; pain in left chest for past 3–4 months; chronic cough at night, with white foaming expectoration but no hemoptysis; weight 108 lbs., weight one year ago 117 lbs.; height 64½ inches.

*Blood.*—Erythrocyte count 3,390,000; hemoglobin estimation 8.5 grams percent; hematocrit 32 percent; corrected sedimentation rate 28 millimeters at the end of one hour; white blood cell count 21,900.

*Urine.*—White blood cells in high-power field 5–10; 0–2 waxy casts.

*Chest film.*—Area of increased density with fluid level, left upper lobe. Further study indicated.

Follow-up revealed patient left employment on February 1, 1951. On May 2, 1951, a thoracotomy was performed on patient, which revealed an infiltrating epidermoid carcinoma of left lung. The patient died on May 27, 1951, with diagnosis of carcinoma of left lung.

*Case No. 5.*—White male, 53 years old. See Figure 20.

*Employment History:*

*Chromate.*—From 1947 to present time patient was a training supervisor and trained workers in potash plant and in liquor building. Most of last year he was in the office.

From 1935 to 1947 he was a mill and roast foreman; one-third of the time in mill rooms and two-thirds of the time equally between kiln and leach areas.

*Nonchromate.*—From 1932 to 1935 patient was foreman for an insecticide company. From 1925 to 1932 he was foreman in a paint company. From 1918 to 1925 he worked as a laboratory technician in insecticide company. From 1917 to 1918 he was in the U. S. Army during World War I. Prior to 1917 he worked in the Baltimore City Health Department bacteriological laboratory.

*Medical History:*

*Habits.*—Two packages of cigarettes daily until June 1, 1950, when he gave up smoking because of a chronic cough.

*Previous Illness.*—Had pneumonia in 1943. The patient has had "chrome ulcers" on hands; however for the last 10 years has not had any new ones.

*Previous Medical Examinations.*—Preplacement and periodic examinations were negative. The patient had his first periodic chest film on May 18, 1948, which showed some accentuation of the root and bronchial markings with exaggerated strands extending into the midzones of the right lung in the first and second interspaces anteriorly. These changes became progressively pronounced up to the third film on June 14, 1949, at which time soft tissue infiltration was demonstrable in the second interspace anteriorly on the right. On the subsequent film (taken in March 1950) there was suggested improvement in the process under observation.

On August 1, 1950, the Public Health Service survey team examined this patient with the following findings: chronic cough (productive with hemoptysis during the past two months); intermittent, mild chest pain on lower right side (not related to exercise or breathing); weight 243 lbs., weight one year ago 226 lbs.; blood pressure 170/120; pharynx hyperemic; excessive crusting of nasal septum but no perforation; chest and abdomen negative.

*Blood.*—Erythrocyte count 5,390,000; hemoglobin estimation 17.5 grams percent; hematocrit 37 percent; corrected sedimentation rate 25 millimeters at the end of one hour; white blood cell count 11,200.

*Urine.*—Fatty casts.

*Chest film.*—Definite enlargement above right hilar area. Further workup needed to rule out bronchiogenic carcinoma.

*Present Illness.*—Subsequent to date of survey the process became more extensive, with retraction of mediastinum to site of pathologic condition and mottled infiltration of the upper third of the right lung. The last three films of the series rather definitely indicate the appearance of a soft tissue mass (about the size of a small "lemon") lying just above the right hilar structures with subsequent atelectatic changes of the right lobe presumably on the basis of partial occlusion of the right bronchus. On April 19, 1951, the impression of the consultant radiologist was that this patient had a bronchiogenic carcinoma, infiltrating the right upper main bronchus.

On January 11, 1951, the patient was examined by the plant's consulting internist who elicited cough and loss of appetite in patient's history, and found rales of different types in the right lung. Fluoroscopy revealed a pathologic condition in upper lobe of right lung. The internist recommended bronchoscopy.

On January 16, 1951, the patient was bronchoscoped with negative findings and was diagnosed as having had bronchitis. Bronchial washings taken at this time were described as, "Some atypia such as nuclear enlargement and multinucleation has been found more in left than in right bronchial specimen. Significance not clear. Neoplastic cells have not been seen, Class II."

On February 27, 1951, the patient refused surgical treatment. He refused any further workup. His course was progressively worse, as seen on subsequent films and by aggravation of symptoms; e.g., fig. 20, May 4, 1951.

The patient died in February 1952; a necropsy was performed.

*Pathological Diagnoses.*—1. Squamous cell bronchiogenic carcinoma with metastasis to striated muscle, adrenal gland, lymph node and lung parenchyma. 2. Congestion of lung, kidney, spleen, and liver. 3. Fatty metamorphosis of liver. 4. Benign prostatic hyperplasia.

The chemical analysis for chromium of various tissues obtained from the necropsy appears in the text table on page 93 of the section, **Pulmonary neoplasms.**

**Case histories — Proved cases**
***(diagnosed as cancer before survey)***

*Case No. 6.*—White male, 63 years old. See Figure 21.

*Employment History:*

*Chromate.*—From March 21, 1928 to 1951 patient was employed as a cooper (put heads on barrels) and helped shipping clerk. Came in contact with finished products of chromates, mainly bichromates.

*Nonchromate.*—He was a cast-iron molder in foundry from 1903–1928, where he did his own shakeout work for the first ten years of employment.

*Medical History:*

*Habits.*—Smoked an average of 14 cigarettes per day.

*Previous Illness.*—In one year in chromate exposure patient developed a perforation of the nasal septum. Had chronic bronchitis from 1920–1922 only. This patient has worked continuously up to present illness with a record of no loss of time in past 20 years.

*Previous Medical Examinations.*—Preplacement and periodic examinations were negative, except for presence of bilateral hernia. On February 9, 1948, patient had his first periodic chest film which was read as negative. On August 9, 1948, patient's periodic chest film was taken and referred to a roentgenologist who, on comparing the two periodic chest films, stated: "There is considerable difference in the quality of the films, making it rather difficult to evaluate the comparison. A number of calcifications are seen clustered at each hilum and along the lung roots, all of which appear stable in character. There is no apparent infiltration of the parenchymal portions of the lungs. On the present film, there is some apparent widening of the aortic shadow, seemingly of both the ascending and descending arches. This latter finding should be further evaluated by fluoroscopy."

At this time the patient had no complaints or other findings.

On August 27, 1948, the patient was fluoroscoped to evaluate the enlarged aortic shadow. The fluoroscopic report was as follows: "There was no fusiform enlargement or apparent pulsating mass. No posterior infringement on esophagus. Oblique views of the chest confirm this impression. Conclusion: General elongation and widening of aorta with no evidence of aneurysm or neoplasm."

On February 22, 1949, the patient had his semi-annual periodic examination which was declared to be negative.

On June 16, 1949, on a routine periodic examination, the patient had a weight loss of three pounds. The plant physician noted that the skin of the patient's abdomen revealed evidence of weight loss. However, no significance was placed on chest film.

*Present Illness.*—On July 16, 1949, patient complained of lassitude and "not feeling just right" and took a vacation for one month. Upon his return his chest was X-rayed again and it was noticed at the time that he had a weight loss of 25 pounds (weight 106 pounds). Because of film changes he was bronchoscoped which revealed some dilation of the bronchi with chronic massive congestion, mainly at the base. One area in right bronchus had a tendency to bleed freely which was suggestive of carcinoma. Bronchial secretions were saved and examined for neoplastic cells, and bronchial washings were obtained. The bronchial secretions revealed pleomorphic changes of desquamated epithelial cells, suggestive of neoplasm. The bronchial washings were reported as "Right and left bronchial washings show conclusive evidence of malignant neoplasm, most likely a bronchiogenic carcinoma. Malignant cells were found in both specimens, Class V."

On August 18, 1949, chest film was repeated, as well as on September 14, 1949, and October 11, 1949, all revealing the mass on right side becoming more extensive. Small masses can be observed now in the second and third interspaces.

On November 10, 1949, periodic examination revealed increase in breath sounds posteriorly. The patient complained for the first time of slight hoarseness and some dyspnea. Weight 126 pounds. The chest film reveals extension of process with atelectatic and inflammatory, as well as pleuritic, changes. Fig. 21 shows chest film of Jan. 16, 1950.

On April 18, 1950, periodic examination revealed patient's weight to be 122 pounds. The patient stated that he had experienced an increase of hoarseness. Chest films taken each month reveal extension of the lesion on the right.

On July 21, 1950, the patient was examined by the Public Health Service survey team and the following findings were recorded: No cough or hoarseness; tinnitus both ears; crusting and perforation of nasal septum; epigastric burning sensation and nocturia; pterygium right eye; blood pressure 186/100; pulse 96 per minute; respirations 24 per minute; temperature 98.4° F.; weight 115 lbs., weight one year ago 128 lbs.; height 64¼ inches.

*Blood.*—Erythrocyte 3,720,000; hemoglobin estimation 12.0 grams percent; hematocrit 40 percent; white blood cell count 15,600; corrected sedimentation rate 33 millimeters at the end of one hour; chromium content in blood 0.00/mg percent.

*Urine.*—Albumin positive 10 milligrams per 100 milliliters; red blood cells per high-power field 20–30; coarse granular casts per high-power field 0–1.

*Chest film.*—Highly suggestive of bronchiogenic carcinoma. Nodules suggestive of silicosis were noted.

On December 1, 1950, patient's weight was noted to be 117 lbs., urine was positive for albumin. At this time patient found it difficult to work at his regular job, and was transferred to do odd jobs about the laboratory. He died November 1951.

*Case No. 7.*—White male, 62 years old. See Figure 22.

*Employment History:*

*Chromate.*—From 1950 to the present time patient worked as a gateman. From 1949 to 1950 he was ill (see *Present Illness*). From 1923 to 1949 he was a furnace man.

*Nonchromate.*—From 1919 to 1923 patient worked as fireman for a sugar company. From 1913 to 1919 he worked as fireman for a railroad.

*Medical History:*

*Habits.*—Smoked an average of 15 cigarettes a day.

*Previous Illness.*—After a year of exposure to chromates patient developed a perforation of the nasal septum. In 1946, patient had pneumonia and was successfully treated with sulfonamides. The patient has enjoyed good health up until present illness.

*Previous Medical Examinations.*—Periodic examinations at the plant were essentially negative.

*Present Illness.*—The patient complained of cough and pain in the chest in November 1949. He was hospitalized, where an initial diagnosis of bronchopneumonia was made. This was later changed to bronchiogenic carcinoma. A thoracotomy was done, and it was found that the patient had a bronchiogenic carcinoma of upper lobe with metastasis. A biopsy revealed squamous cell carcinoma. He convalesced for a year and then returned to work for the chromate plant. Fig. 22 shows chest film of Dec. 8, 1949.

On October 31, 1950, the Public Health Service survey team examined

this patient and the pertinent findings are as follows: Large nasal perforation with streaking; dyspnea, mild, with constant cough for 1½ years (with frothy white expectoration); anorexia for the past year; thin in appearance, weight 134 lbs., weight one year ago 165 lbs.; blood pressure 124/72; perforated and retracted right ear drum.

*Blood.*—Erythrocyte count 4,890,000; hemoglobin estimation 15 grams percent; hematocrit 38 percent; corrected sedimentation rate 22 millimeters at the end of one hour; white blood cell count 10,600.

*Urine.*—Negative.

*Chest examination.*—Wheezing and rhonchi on expiration in right upper lung fields, antero-laterally and posteriorly. Enlarged axillary and inguinal lymph nodes.

*Chest film.*—Hilus enlarged; dense mass right upper lobe. Impression—bronchiogenic carcinoma.

Three weeks after this examination the patient was found dead in locker room of plant. Immediate cause of death was gross hemorrhage. No necropsy could be obtained.

*Case No. 8.*—White male, 53 years old. See Figure 23.

*Employment History:*

*Chromate.*—From 1950 to present time patient was foreman of entire plant. From January 1950 to June 1950 he was ill. From 1941 to 1950 he worked as foreman. From 1938 to 1941 he was watchman. From 1929 to 1938 he worked as a laborer. For 3 months in 1929 he was an oiler.

*Nonchromate.*—From 1927 to 1929 patient worked on assembly line of a motor car company. From 1921 to 1927 he was an expressman. From 1920 to 1921 he worked as brakeman for railroad. From 1916 to 1920 he was in the U. S. Army. Prior to 1916 he was in school.

*Medical History:*

*Habits.*—Smoked an average of 10 cigarettes a day.

*Previous Illness.*—When first employed in chromate plant, patient had several chrome ulcers which have since healed.

*Previous Medical Examinations.*—Periodic examinations at plant have not been significant. Fig. 23 shows chest film of Feb. 4, 1950.

*Present Illness.*—Patient was hospitalized on November 19, 1949, with diagnosis of pneumonia. However, suspicion of malignancy was aroused; he was bronchoscoped and the biopsy revealed bronchiogenic carcinoma of right lung.

He had a pneumonectomy in February 1950. He returned to work in June, 1950.

On October 21, 1950, Public Health Service survey team examination of this patient revealed: Dyspnea; productive cough for the past 1½ years with about 1 tablespoonful of white sputum 4 times a day; gnawing pain in left epigastric region with intermittent distress; chronic constipation since February 1950; congestion of conjunctiva; crusting of nasal septal mucosa but no perforation; healed chrome scars on hands; breath sounds only on left side of chest.

*Blood.*—Erythrocyte count 4,850,000; hemoglobin estimation 15 grams percent; hematocrit 39 percent; corrected sedimentation rate 16 millimeters at the end of one hour; white blood cell count 7,500.

*Urine.*—Negative.

His family physician as of June 27, 1951, reports him to be apparently well.

**Case histories — Radiological evidence only**
***(not diagnosed as cancer at time of survey)***

*Case No. 9.*—Colored male, 48 years old. See Figure 24.

*Employment History:*

*Chromate.*—From 1942 to present patient was a barrow man in the kiln room.

*Nonchromate.*—From 1940 to 1942 patient worked in a company where wax for polishing cars was made. From 1930 to 1940 he worked at odd jobs (laundry worker and laborer). From 1928 to 1930 he was a laborer in a shipyard. Prior to 1928 he farmed.

*Medical History:*

*Habits.*—Smoked an average of 10 cigarettes a day and used 2 plugs of chewing tobacco.

*Previous Illness.*—The patient had pleurisy in 1915. After 4 months' exposure to chromates he developed a perforated nasal septum. On May 7–19, 1939, he was hospitalized for acute pharyngitis, acute glomerulonephritis, and urethral stricture. He was hospitalized on March 7–19, 1942, for bronchopneumonia. During his stay a 1–1000 tuberculin test was positive but two sputum examinations were negative. On January 30, 1948, as an out-patient he was diagnosed as having chronic bronchitis, and arthritis of lower thoracic and upper lumbar vertebrae. At that time his chest film was read as "There are numerous rounded opacities in both lungs posteriorly at bases. These have a bronchiogenic distribution. There is no evidence of patent tuberculous lesion, intrathoracic calcification or mediastinal adenopathy. Chronic pleural changes are seen at right base with blunting of costophrenic sinuses and pleural thickening along lateral chest wall. Impression: Findings compatible with tuberculosis or fungous infection." On February 18, 1948, the patient complained of cough with moderate expectoration. Chest film taken at the time was read as, "Scattered fibrocalcific infiltration throughout the left lung with more fibrosis in the left upper third. There is similar infiltration to a lesser extent in the right lung and there is an old pleurisy obliterating costopleuric angle. Impression: Moderately advanced pulmonary tuberculosis, probably inactive." On June 14, 1950, patient was reported to have had chrome ulcers on hands, which gradually healed.

*Previous Medical Examinations.*—Preplacement physical and periodic examinations were negative except for nasal perforation.

On July 14, 1950, the Public Health Service survey team examined the patient and found the following significant findings: Crusting and perforation of nasal septum; no chest pain, hoarseness, cough or hemoptysis; weight 154 lbs., weight one year ago 161 lbs.; blood pressure 100/60; chrome scars on hands; chest and abdomen negative.

*Blood.*—Erythrocyte count 5,250,000; hemoglobin estimation 17.5 grams percent; hematocrit 39 percent; corrected sedimentation rate 35 millimeters at end of one hour; white blood cell count 4,100.

*Urine.*—Negative.

*Chest film.*—Scattered calcific nodules, increased left hilar area. Further study needed.

*Present Illness.*—In January 1951 patient developed pain in left shoulder which spread anteriorly to the posterior portion of the chest and was aggravated on deep inspiration. However, this pain disappeared. Patient lost 35 lbs. in weight and became progressively weaker so that he was not able to work. He was admitted to a hospital on June 11, 1951, with the chief complaint of weakness, loss of weight and pain in left anterior chest. His other symptoms were anorexia, and intermittent dysphagia. However, he did not have any

dyspnea, palpitation, cough or hemoptysis. On physical examination he appeared cachectic, weak and chronically ill. Nasal perforation was noted but otherwise eyes, ears, nose and throat were negative. Chest examination revealed diminished breath sounds over the left upper chest posteriorly and vocal fremitus was diminished. There was slight limitation of respiratory excursion bilaterally. There was no dullness or hyperresonance to percussion. Impression at this time was that of a malignancy of the stomach or esophagus. On June 14, 1951, the patient had an esophagram which was read as, "Fluoroscopically, the barium column was held up just below the level of the aortic arch, but soon passed beyond this point, revealing a somewhat fusiformly narrowed esophagus for a distance of approximately 11.5 cm. This narrowing was on one side of the esophagus only, and was slightly irregular, but not grossly as one sees in carcinoma of the esophagus. The other side of the barium column was entirely smooth in appearance. The very proximal portion of the esophagus, just below the pharynx, was narrowed fluoroscopically but revealed adequate dilation, both fluoroscopically and on the films."

The chest film taken on this same date (June 14, 1951) was read as, "The film reveals a large mass in the left hilus, with slight deviation of the barium column at the right at the point of the mass. The mass exactly corresponds to the area of narrowing of the esophagus reported above. There are numerous small calcifications dispersed throughout both lung fields, doubtless representing healed lesions of a fungous infection, perhaps histoplasmosis. There is also suggestion of fibrosis at the apices. (See fig. 24.)

"*Impression.*—Carcinoma of the bronchus, left, with fairly marked extrinsic pressure upon the esophagus as revealed in the esophagram. Old, healed fungous infection of the lungs. Emphysema, bilaterally. Probable apical tuberculosis, probably inactive." On June 14, 1951, patient was discharged with a diagnosis of bronchiogenic carcinoma, inoperable. Three weeks later he died at home; no necropsy was obtained.

*Case No. 10.*—White male, 54 years old. See Figure 25.

*Employment History:*

*Chromate.*—From January 1, 1951 to January 11, 1951 patient worked as a laborer loading and unloading soda ash. From 1949 to 1951 he was a laborer with yard gang. From 1918 to 1949 he was fireman in furnace room where ore was roasted.

*Nonchromate.*—From 1916 to 1918 patient was a laborer in a railroad power house. From 1911 to 1916 he worked with a railroad section gang. Prior to that he farmed.

*Medical History:*

*Habits.*—Smoked only one cigarette a day.

*Previous Illness.*—None offered.

*Previous Medical Examinations.*—Patient developed perforation of the nasal septum after 2 years of exposure to chromates. Preplacement and periodic physical examinations have otherwise been negative.

On January 11, 1951, the Public Health Service survey team examined this patient and recorded the following: Productive cough during last winter and constant for the last 3-4 months, expectoration frothy white, approximately 2 3 tablespoonfuls, 5 6 times a day; no hemoptysis or chest pain; crusting, whistling and large perforation of nasal septum.

*Blood.*—Erythrocyte count, white blood cell count and hemoglobin estimation not done as patient would not submit to finger puncture; corrected sedimentation rate 28 millimeters at the end of one hour; hematocrit 40 percent.

*Urine.*—Negative.

*Chest film.*—On right, enlargement of lung root with area of increased density extending out from this region into the upper portion of lower third; left lung field clear; heart and aorta normal. Compatible with roentgenological diagnosis of bronchiogenic carcinoma. (See fig. 25.)

On July 15, 1951, patient complained of extreme lassitude and fatigue to his family physician, who hospitalized him. He was found to have a red blood cell count of 1,700,000 and extension of chest lesion on the right. He was given blood transfusions and treated with antibiotics for pneumonia. The patient's red blood cell count was restored to 4,500,000 and he felt symptomatically well. However, the lesion in the right lung field failed to clear up and the patient is under observation at the present time as a bronchiogenic carcinoma case.

**Proved old case (former chromate worker)**

*This case is presented for several reasons. It illustrates how people who have worked in the chromate industry for a considerable period of time may devlop bronchiogenic carcinoma even though they have changed employment. By this change of jobs the occupational history may fail to record exposure to chromates, as in the case of this patient where no mention of his employment in a chromate-producing plant was made on the hospital records. Colored male, 48 years old.*

*Employment History:*

*Chromate.*—From 1931 to 1945 patient was a laborer in one chromate-producing plant.

*Nonchromate.*—Prior to 1931 patient did odd laboring jobs, drove a truck, and farmed. Since 1945 he has been a preacher.

*Medical History:*

*Habits.*—Smoked an average of 20 cigarettes weekly.

*Previous Illness.*—Usual childhood diseases. Influenza in 1918.

*Previous Medical Examinations.*—Periodic examinations did not reveal any significant findings.

*Present Illness (First Admission).*—This patient was admitted to a government hospital on June 24, 1947, complaining of chronic cough and shortness of breath. Three months prior to admission he developed a chest cold which lasted about one week but which persisted in the form of a dry, nonproductive hacking cough. At time of admission the cough was worse in the early morning and late at night. A negative chest film had been taken by a private physician. Three weeks before admission he first noticed shortness of breath on climbing steps or a hill, but not on level ground. He had no orthopnea or peripheral edema, but had had nocturia four to five times at night.

*Physical findings:* Thrill palpable over the sternum, more marked in the second right interspace; P.M.I. was in the 6th left interspace, 12 cm from the midsternal line; blowing systolic murmur in the mitral area and a harsh, rough, systolic murmur in the second right interspace, as well as a diminuendo diastolic murmur in the same area; dullness on percussion at the left base posteriorly; breath sounds were decreased and whispered noise sounds were poorly transmitted in the same area.

*Laboratory findings.*—Erythrocyte count 3,970,000; hemoglobin estimation 12.5 grams percent; hematocrit 33 percent; white blood cell count 6,450; Kline positive, Kahn 4 plus with 64 Kahn units; spinal fluid 180 cells per cubic meter; slight trace of globulin; 15 milligrams percent total protein; negative quantitative Kolmer Wassermann.

*Chest X-ray.*—The chest film taken on admission was read as, "The lungs show considerable density involving most of the lower left lung. It is somewhat

demarcated. The left dome of the diaphragm is elevated and this area is obscured by the lung changes. The left upper lobe and right lung appear quite clear. The heart shadow shows evidence of enlargement. The left border is obscured by the changes in the left lung. The aorta shows considerable dilation of the ascending section. A fusiform type aneurysm may be developing."

The next chest film was obtained on July 3, 1947, which was read as, "No definite change identified in the lower left lung since the previous examination. The entire lower section of the left lung field is still completely obscured with the left dome of the diaphragm elevated. These changes are known to be mostly in the posterior section of the lung. The lung field from the hilum downward and outward is almost completely obscured." The patient was given bismuth therapy for his aortitis and sulfonamides for his febrile reaction secondary to collapse of lower left lung.

On July 14, 1947, patient had another chest film which revealed that the entire left lung field, except for the apical area, was quite dense. This density was believed to be greater than that expected in atelectasis, hence fluid was suspected. No change was noted in the entire right lung. The chest film of July 17, 1947, shows the left lung field to be less obscured than previous film. There is a general haziness up to the level of the clavicle with marked density seen from the lung root region to the diaphragm, which is markedly elevated. The chest film of July 17, 1947, shows no new changes.

On July 22, 1947, the patient was bronchoscoped which revealed narrowing of left main bronchus. No ulceration or areas of nodularity were noted. Biopsies were taken in the area of constriction, the report being "no tumor found."

On August 2, 1947, chest aspiration was done. Smear and clot showed adult lymphocytes and a few pale mononuclear cells. Red blood cells were present. No tumor cells however were identified.

The last chest film July 28, 1947, before the patient's discharge revealed essentially greater density and some progression of the density in left lung field.

The patient was discharged on August 16, 1947, with the main diagnosis of marked aortitis with regurgitation. It was further added that the constriction of the left lower main bronchus was due to an aneurysm of the descending aorta rather than a primary growth of the bronchus. It was decided that, regardless of whether there was a primary carcinoma of the bronchus or not, thoracotomy would be inadvisable because of the patient's general condition.

*Second Admission October 23, 1947.*—On admission it was learned that the patient had felt quite well until one month before admission when he developed hoarseness and weakness. Two weeks before admission he developed a slight cough and for one week had coughed up varying amounts of clotted and bright red blood. He was moderately dyspneic on exertion and had nocturia for the past two weeks.

Physical examination revealed a dyspneic, acutely ill male, coughing blood. His entire left chest was flat on percussion. An occasional faint breath sound was heard posteriorly on the left. Right chest showed many coarse rales, especially at the base, posteriorly. Heart was displaced to the right side. A thrill was palpated over the second right interspace. Aortic diastolic and systolic murmurs were heard. Blood pressure was 160/100. The abdomen was distended and tense. There was no peripheral edema.

*Chest X-ray.*—This chest film revealed that the entire left lung field was

as dense as the heart shadow. The heart and mediastinal structures were displaced to the right. These changes indicated the presence of a large pleural effusion.

On October 28, 1947, the chest was aspirated of 400 cc of bloody fluid and a smear of the centrifuged sediment revealed numerous large abnormal "pavement-like" cells with eosinophilic cytoplasm and hyperchromatic nuclei; positive for neoplastic cells.

On November 5, 1947, left cervical lymph nodes were biopsied revealing closely packed cords and strands and sheets of tumor cells. Nuclei were large, oval and stained faintly basophilic, cytoplasm was scant. Bronchoscopy was repeated with negative results. Again surgical intervention was not deemed advisable. The patient was given X-ray therapy to the left cervical region and was discharged on December 2, 1947.

*Third Admission December 16, 1947.*—Patient was readmitted two weeks after previous discharge in severe cardiac failure. Physical examination revealed bilaterally distended neck veins, marked dyspnea, orthopnea and ankle edema. Liver was enlarged and tender. The entire left chest was dull to percussion with absence of voice and breath sounds. The heart was pushed to the right with the right border of cardiac dullness 4 cm to the right of the sternal border. Pulse was 130 per minute and irregular. Eighteen hundred cc of bloody fluid was aspirated from the left chest. An EKG showed left axis deviation, low voltage in all leads, a ventricular rate of 201. X-ray examination of the chest at this time showed the heart and mediastinum shifted to within 3 cm of the right wall. The entire left chest was obscured by fluid. The patient became gradually weaker and more dyspneic. His blood pressure as recorded dropped to 76/0 and his heart beat increased to approximately 200 per minute. He died on December 17, 1947.

*Necropsy Protocol.*—Because necropsy material on bronchiogenic carcinoma in chromate workers is scanty, the protocol is reviewed completely.

*Microscopic Examination of Lungs and Tumor.*—A longitudinal section through the bifurcation of the right main bronchus shows marked replacement of the mucosa and submucosa with almost complete destruction and necrosis of these layers by irregular sheets of densely packed hyperchromatic neoplastic cells. The area around the bronchus is likewise infiltrated, replaced, and destroyed. Here the neoplastic cells can be seen in vascular spaces and there are large areas of necrosis. Extension to the lumina of adjacent alveoli with fibrotic and degenerative changes in their walls can be seen. Neoplastic cells have a moderately abundant fibro-collagenous stroma composed of thick cords of collagen dividing the masses of cells into smaller islands and finer strands passing among smaller groups of cells. The stroma is relatively avascular. The individual neoplastic cells have scant cytoplasm. The majority are stellate in shape and have fine protoplasmic processes. In both areas these cells are so closely packed together that the cell outlines are lost. The nuclei are small, round to oval, and hyperchromatic. No nucleoli are seen. Mitoses are relatively numerous. Other areas of the lung show extensive involvement of groups of alveoli with complete filling of the lumina and involvement of the lymphatics of the alveolar tubules as well as those of the bronchioles. Large areas of degeneration and necrosis are again seen as well as focal areas of hemorrhage within the tumor. Occasionally neoplastic cells are seen within the lumina of small bronchioles. The uninvolved areas of lung are relatively normal except for scattered numerous intra-alveolar macrophages or desquamated lining cells laden with black pigment granules.

In some areas the appearance of the tumor suggests a papillary growth around a connective tissue core. This appearance is rare.

*Gross Necropsy of Lungs.*—The right lung weighs 520 grams. Except for the adhesions noted at the apex no other changes are seen in color or consistence externally. On cut surface the lung is a normal salmon pink color flecked with areas of anthracosis. There was no evidence of congestion, edema, or pneumonia. The bronchi and pulmonary arteries are patent. The left lung together with the large masses of mediastinal and pericardial nodes weighs 1,400 grams. The trachea is divided in the mid-line posteriorly and is normal down to and including the carina. The left lung is reduced to about one-eighth of its usual volume and except at the apex it is of the consistence of liver. It does not contain air. The most severely involved portion appears to be that area around the main bronchus at its bifurcation. Nothing remains of the upper lobe bronchus. The tumor has infiltrated densely and is firm, white, and fibrous. It has extended to involve the adjacent pleura and pericardium as well as the pericardial pleura of the left upper chest. Many of the bronchioles below the point of obstruction by tumor contain plugs of thick white mucous. Others contain greenish-yellow purulent material.

*Final Diagnosis.*—Bronchiogenic carcinoma of the left main bronchus with extension to right lung, pleura, parietal and visceral pericardium, and the regional lymph nodes and metastases to heart, liver, periaortic lymph nodes, mediastinal lymph nodes, cervical lymph nodes, and mesentery resulting in hemopericardium, atelectasis of left lung and hydro-hemothorax, left; syphilitic aortitis with marked dilatation.

## *Epidemiological considerations*

Lung cancer found by the medical examination of chromate workers is without doubt much more common than would usually be observed among other populations. Nevertheless, it is worthwhile to attempt measuring the difference. For purposes of comparison, the prevalence of lung cancer among persons in the general population is needed. Most studies of cancer prevalence are not comparable since they deal with hospital cases or cases seen by physicians. Such persons already knew they were ill and were seeking medical treatment. Mass chest X-ray surveys cover a fairly representative group of the entire population, but usually the emphasis has been on tuberculosis rather than on cancer detection. However, an X-ray survey involving 536,012 persons was conducted in Boston during 1949–50, at which time a special effort was made to detect lung cancer cases *(111)*. All cancer suspects were given a thorough examination including a large X-ray film, with the result that 76 cases, or 14.2 per 100,000, were diagnosed as cancer. Of these, 43 were proved to have primary cancer of the lung; three were other types of malignancy; fifteen had metastatic lesions to the lung from other parts of the body; the remaining 15 had presumptive evidence indicating cancer, but the diagnosis was not conclusive. According to sex, 54 were males and 22 were females.

Table 41 shows prevalence rates of bronchiogenic cancer among chromate workers compared with rates for males diagnosed as lung cancer in the Boston chest X-ray survey.

When all cases which might possibly be classified as lung cancer in the Boston survey are compared with bronchiogenic cancer proved or diagnosed clinically and radiologically in the chromate industry, the rate per 100,000 for the former is only 20.8 while the rate for the latter is 1,115. Considering the age group 15–69 years, if the diagnosed cases are excluded from the chromate calculation and prevalence is based on eight proved cases, but all diagnosed cases in the Boston survey are retained, the difference is still tremendous. The rates are now 892 and 18.6 per 100,000, respectively. If these data are further refined by including only those five proved chromate cases which were unknown prior to the survey, the rate among chromate workers falls to 557.

Comparison of rates by age shows the greatest excess for chromate workers 40–49 years old. Whereas the bronchiogenic cancer rate for chromate workers is 32 times the Boston lung cancer rate at age 60–69 years, it is 325 times that for the Boston males at age 40–49 years. This strongly suggests that some factor not found in Boston is producing bronchiogenic cancer cases more frequently and at an earlier age among chromate workers.

TABLE 41.—*Comparison of bronchiogenic cancer among male chromate workers and lung cancer among males examined in a chest X-ray survey in Boston, according to age.*

| Age group (years) | Chromate workers | | | Boston chest X-ray survey | | |
|---|---|---|---|---|---|---|
| | No. of workers | Cancer cases | Rate per 100,000 population | No. of persons surveyed | Cancer cases | Rate per 100,000 population |
| All ages | 897 | 10 | 1,115 | 259,072 | 54 | 20.8 |
| 15–19 | 1 | 0 | 0 | 26,859 | 1 | 3.7 |
| 20–29 | 129 | 0 | 0 | 70,142 | 0 | 0 |
| 30–39 | 250 | 0 | 0 | 56,264 | 3 | 5.3 |
| 40–49 | 210 | 3 | 1,429 | 45,697 | 2 | 4.4 |
| 50–59 | 195 | 3 | 1,538 | 34,409 | 20 | 58.1 |
| 60–69 | 112 | 4 | 3,571 | 19,023 | 21 | 110.4 |
| 70 or over | 0 | – | – | 6,678 | 7 | 104.8 |

# Summary and Recommendations

## Summary

From the six chromate-producing plants, employing about 935 persons, 897 males were medically examined.

Perforation of the nasal septum was found in 509, or 56.7 percent, of these chromate workers. It was noticed that the colored chromate workers had a greater prevalence of nasal perforations than the white chromate workers. They also developed their perforation in a shorter period of exposure to chromates, as 42.3 percent of the white chromate workers experienced nasal perforations within the first year, compared to 65.7 percent of the colored chromate workers. A study of 87 chromate workers revealed a very strong relationship between the absence of perforation and the practice of prophylaxis.

The most common eye finding was congestion of the conjunctiva which was found in 44.7 percent of the white and 22.5 percent of the colored chromate workers, compared with 35.1 and 17.3 percent for white and colored nonchromate workers, respectively. It was found that none of the ophthalmological symptoms or findings in chromate workers increases with age, which suggests that the irritation of the eye is limited and is not aggravated by length of employment.

Severely red throats were found in 9.8 percent of white and in 12.8 percent of the colored chromate workers, but in only 1.4 percent of white and 7.4 percent of colored nonchromate workers.

There was no evidence to show that exposure to chrome compounds affected the rate of dental caries attack. Some of the workers, however, developed a yellowish discoloration of the teeth and tongue. A higher percentage of the chromate workers experienced gingivitis and periodontitis.

Chromate workers exhibited vital capacity levels above that found in a group where pulmonary fibrosis was higher than normal.

Pulmonary markings suggestive of fibrosis were not important among chromate workers but bilateral hilar enlargements were observed. The severity of tuberculosis was less among the chromate workers than in another observed population.

Neither occupation nor exposure to chromates appears to influence the prevalence of hypertension or other cardiovascular diseases.

A median sedimentation rate of 11.3 mm was obtained for all chromate workers.

White and red blood cells and casts in urine appeared more frequently than is usually observed in the average industrial population. These findings tend to increase with increasing years of exposure in the chromate-producing industry.

Coproporphyrins were within the normal limits of 100–120 micrograms per 24 hours.

In a selected study of cases in which bronchiogenic carcinoma was suspected, mucoprotein and polysaccharide levels were elevated in nearly one-third of the cases. Three definitely known cases of bronchiogenic carcinoma showed high levels.

Of the 897 chromate workers examined 10 were considered as having bronchiogenic carcinoma. Of these, eight had, in addition to roentgenological and clinical findings, histological evidence (pneumonectomy in 5 cases, biopsy in 1 case, necropsy in 1 case and bronchial washings Class V in 1 case). The other two had, in addition to a suspicious film taken on the survey, extension of the lesion on subsequent films and clinical findings of bronchiogenic carcinoma. The mean age of these 10 workers was 54.5 years and the mean exposure to chromate 22.8 years.

A survey of a comparison group showed 20.8 lung cancer cases per 100,000 people, whereas the rate for bronchiogenic cancer among chromate

workers was 1,115. In considering only chromate cases proved by histological findings but all diagnosed cases in the comparison group, for age group 15 to 69 years, the rates were 892 and 18.6 per 100,000, respectively. If one includes only cases which were unknown prior to the survey, the rate among chromate workers was 557 per 100,000. Examination of rates by age showed the greatest excess for chromate workers 40–49 years old (325 times that for this age group in the comparison group). Some factor, not present in the comparison group, is responsible for the greater prevalence and earlier production of bronchiogenic carcinoma in chromate workers.

**Recommendations**

Continue the periodic X-raying of all workers especially those who have worked 5 years or more in the chromate-producing industry. These workers should be X-rayed every 3 months. All X-rays should be read by a competent roentgenologist.

Continue to follow the mortality and morbidity experience of workers employed in chromate plants.

The local health department should follow-up all chromate workers who have worked in the industry 5 years or more.

## DISCUSSION

Evidence has been presented showing an abnormally high prevalence of pulmonary neoplasms among workers employed in plants producing chromates. Because of this, it is appropriate to consider the factors in the working environment which might be etiological.

The basic operations, such as crushing, grinding, roasting, leaching, granulating, neutralizing, filtering, concentrating, drying, and packing are all common to many industries, and there is no evidence that these operations, as such, have any bearing on the question. It appears reasonable, therefore, to surmise that the high prevalence of pulmonary neoplasms may be related to exposure to some substance, or substances, characteristic of this industry. The primary raw materials utilized in the industry are chromite ore, soda ash, lime, sulfuric acid, fuel oil, water and air. Except for the chromite ore all of these substances are encountered in many other industries which have, as far as is known, no unusual prevalence of pulmonary carcinoma.

Consequently, one must consider chromite ore and its derivatives as being primarily responsible for this high prevalence of pulmonary neoplasms in these workers. Most investigators of this problem have reached the same conclusion and there appears to be general agreement that chromium alone or with one or more of its compounds is definitely implicated.

Metallic chromium, as well as hexavalent and trivalent chromium compounds, has been considered the etiologic factor in the production of neoplasms. Schinz *(108)* reported he implanted metallic chromium in the thighs of rabbits and was able to produce sarcomas four years later. In 1932, Lehmann *(66)* exposed animals to a spray of bichromate and although he was able to produce septal perforations, no mention is made of any pulmonary neoplasms. In 1934, Akatsuka and Fairhall *(41)* did not report any neoplasms in animals after the ingestion and inhalation of chromium carbonate and chromic phosphate. Alwens *(70)* reported in 1938 that Gross was unable to produce bronchial carcinoma in animals after inhalation of chrome dust over long periods of time, although the hilar glands revealed hyperplasia. In 1910, Shimkin *(107)* injected chromite ore intravenously into cancer susceptible mice but did not find any increase of neoplasms as compared with the controls. Thus, except for the work of Schinz, none of the animal experiments has succeeded in producing neoplasms from chromium compounds.

As the result of field studies of chromate workers, various investigators considered hexavalent and trivalent chromium compounds as etiologic

agents for the production of pulmonary neoplasms. In 1937, Bauer *(105)* believed that free chromic acid or the alkali salts, especially the bichromates, were responsible for the production of pulmonary neoplasms.

In 1938, Koelsch *(76)* believed the monochromates and bichromates were the responsible agents in pulmonary carcinoma and believed further that the chromites were not harmful, since they are scarcely soluble in the body. In 1939, Gross *(72)* considered the monochromates to be the compounds responsible for the lung neoplasms in chromate workers. Gross and Koelsch *(73)*, in 1943, believed that zinc chromate among pigment workers was the cause of lung cancer because it was more soluble than barium or lead chromate.

Machle and Gregorius *(35)* in 1948, considered the monochromates to be the compounds responsible for lung cancer. However, recently Mancuso and Hueper *(78)* considered chromite ore as a potential carcinogenic agent, and further added to this consideration chromium pigments and chromium alloys. They believed that the insoluble chromium compounds are retained in the lung over long periods of time and may give rise to pneumoconiotic changes.

Thus, the advocates of the hexavalent chromium compounds have stressed their chemical and physiological activity, minimizing their extreme solubility and consequent rapid dissipation. The advocates of the trivalent chromium compounds have stressed their prolonged retention, minimizing their possible lack of physiological activity, due to their extreme insolubility.

The present report proposes to introduce for consideration derivatives of chromite ore, other than those which have heretofore been incriminated in the etiology of pulmonary neoplasms among chromate workers. In the beginning phase of chromate production the chromium is contained in the ore as a trivalent oxide, together with, principally, the oxides of iron, aluminum and magnesium. The ore is very inert chemically, being virtually insoluble in either water or acid. Because of this, the ore can be broken down only by fusion with alkaline materials.

In the roasting process most of the chromium is converted to sodium chromate which is water soluble. Acidification is employed to convert the chromate to bichromate and to chromic acid, both of which are also very soluble in water. However, as pointed out earlier in this report some of the chromite is not converted to the chromate, but is altered due to replacement of iron by alkali metals, and where lime is used, into a calcium chromate-chromite complex. These substances are not water soluble but are soluble in acid. Therefore, in the various stages of chromate production, chromium may occur as (1) insoluble in water or acid, (2) water and acid soluble, and (3) as acid soluble but water insoluble compounds.

Thus, although insoluble chromite ore is converted to water soluble chromate compounds during the roasting process, some of the ore is only partially altered through the replacement of the iron by alkali metals such as

sodium, calcium, or magnesium. This altered material is not water soluble but is far less inert than the original ore and is largely soluble in acid. When lime, as well as soda ash, is employed in chromate production, calcium chromate and calcium chromite may be produced. The calcium chromate is unstable and at roast temperatures may give up some of its oxygen to form a calcium chromate-chromite complex. This complex, while not soluble in water, is soluble in acid.

These acid soluble compounds are produced in the roast and remain principally in the residue. Consequently, they are present in appreciable amounts wherever roast or residue is encountered, which would include most of the dry end of chromate production. Where ore is crushed these acid soluble compounds would not be present. However, where residue is crushed and recirculated, these acid soluble-water insoluble compounds are encountered.

A small amount of the residue is suspended in the leach liquor and thus enters the wet-end processes. Additional residue and roast reach the wet-end portion of the plant as airborne dust. These two sources together with basic chromium sulfate, in some instances, probably account for such acid soluble-water insoluble chromium, as was found in the work room air in the wet end of the plants. It appears likely that most of the residue carried over with the leach liquor is removed with the sodium sulfate. Acid soluble-water insoluble chromium originating from the subsequent processing is predominantly basic chromium sulfate.

The following data from this study and other pertinent information suggest that the pulmonary carcinoma found in this industry is associated primarily with the roast and residue materials.

(1) Sufficient work history data were obtainable for 10 cancer cases to correlate them with their occupational environment. Of the 10 cases, 7 (Nos. 1, 3, 5, 7, 8, 9, 10) had work histories that indicate prolonged exposure to appreciable amounts of the acid soluble-water insoluble form of chromium found in roast and residue. One case (No. 2) had a work history which indicated exposure to chromic acid, sodium bichromate, and acid soluble-water insoluble chromium, but not to roast or residue. Two of the 10 cases had work histories in which the sole occupation was "kiln operator" or "kiln building laborer."

(2) High concentrations of chromite ore are present in the beginning of the process and are present in diminishing quantity throughout the process, with relatively very small quantities occurring in the wet-end processes. Two of the ten cancer cases (Nos. 5, 8) have work histories showing that part of their exposure was to these high concentrations of chromite ore. No cases occurred for men with work histories showing prolonged exposure to high concentrations of chromite ore.

(3) All of the cancer cases were exposed to hexavalent chromium.

This is to be expected since this form of chromium is distributed throughout the plants.

(4) The recent report of Bidstrup *(75)* indicated no unusual incidence of pulmonary carcinoma in workers presently employed in the British chromate industry. It is of interest that this report calls attention to the fact that British operating procedure differs from American and German practice in one important respect. Bidstrup states, "In most factories in the United States of America and in Germany, roasting of the ore is done in two stages. In the second stage, residue from the leaching tanks is dried and crushed and used as a filler in place of limestone. This residue contains as much as one percent of monochromate. In the factories in Great Britain *the residue from the leaching tanks is discarded in a moist condition and carried to dumps outside the factory*." (Italics supplied.)

The causative agent of pulmonary carcinoma in this industry has not yet been determined. Most investigators believe that chromium in some form is implicated. Animal experiments have thus far given negative results for the usual trivalent and hexavalent forms, but these still cannot be ruled out, since it is possible that these in combination with other factors might yield positive data.

Attention is directed to the acid soluble-water insoluble chromium compounds in the consideration of etiologic agents of bronchiogenic carcinoma in the chromate-producing industry. In this connection it must be remembered that the dichromates and monochromates may be reduced in the body to acid soluble-water insoluble compounds which must be considered as a pertinent factor in this problem.

Further biochemical and toxicologic research on the effects of all chromium compounds is indicated.

# APPENDIX

## METHODS OF CHEMICAL ANALYSES

Samples of air-borne particulate materials were collected by four methods: (1) the standard impinger, (2) the electrostatic precipitator, (3) a high volume filter paper sampler, and (4) the filter pads of respirators. Grab samples of settled dusts, ores, roasts and mixes were also taken for analysis. Blood and urine specimens were collected from workmen for correlation of analytical results with the clinical and environmental findings.

Chromium, lead, zinc, sodium, potassium, calcium, fluoride, sulfate, and pH were determined on various samples. In most instances, the determination of chromium was divided into water soluble, acid soluble-water insoluble, and water insoluble-acid insoluble compounds of chromium. These designations refer to the conditions used for the separations as will be described. The results are referred to as water soluble chromium, acid soluble chromium and insoluble chromium.

The selection of procedures for the preliminary treatment of samples and the final determinations of the desired constituents depended upon the type of sample, the combination of constituents to be determined and the anticipated concentrations. The same methods of determination were used on several types of samples. Because of these inter-related factors the description of the analytical procedures has been divided into two parts. In the first part, the preliminary treatments and preparations of the samples for the determinations are given. The second part deals with the methods and procedures used for the actual measurement of the desired constituents.

## *Preparation of Samples*

### Electrostatic precipitator samples

#### *Total weight of sample (1)*

The caps of the collection tubes were removed and any loose dust was returned to the tube. The outside surface of the tube was wiped with a cloth moistened with alcohol then dried with a piece of clean gauze. After standing for five minutes in the balance room the tube and sample were weighed. The sample was rinsed with water into a Phillips beaker. Any adhering dust was loosened by scrubbing with a rubber disc policeman, which fitted snugly into the tube. After removal of all the sample, the tube was rinsed with alcohol which was discarded. The tube was wiped inside and out with an alcohol dampened cloth, dried with gauze and, after five minutes standing, weighed. The weight of sample was then obtained by difference.

The tubes tend to pick up moisture from the air or any direct contact with the hands. After the alcohol-wipe and drying, they were handled with clean, dry gauze pads until weighed. The period of standing between wiping and weighing was the same for both weighings of a tube. Five minutes

has been found to be an adequate period although a time equilibrium with the ambient air is not established then. Both weighings were completed within a reasonable time as changes in the humidity affect the results.

### *Chromium*

After being rinsed from the collection tube, the sample was allowed to stand at room temperature for 30 minutes before filtering through paper. This filtrate was analyzed for chromium by the alkaline peroxide procedure (Procedure I) and the results reported as total water soluble chromium (including both hexavalent and trivalent chromium compounds).

The residue and the filter paper were returned to the original beaker. After the addition of 3 ml of concentrated nitric acid, the mixture was warmed and agitated until the paper was well macerated. Then 10 ml of water was added and the mixture heated below boiling for 15 minutes. The heating was continued for 15 to 30 minutes longer after the addition of 25 ml of water. This solution was filtered on paper and washed with water. The combined washings and filtrate were analyzed for chromium by Procedure I. This chromium was reported as chromium from acid soluble-water insoluble compounds.

The filter paper and residue from the acid treatment were ashed in a muffle furnace at 550° C. Chromium in the ash was determined by a fusion method (Procedure II).

### *Lead*

The determination of lead was required on some of the samples treated for chromium as previously described. In these instances, composite samples were made by combining equal fractional aliquots of the water soluble, acid soluble, and acid insoluble portions of the samples. Any residue from the water leaching of the fusion was dissolved in nitric acid, made to volume and a proportional aliquot taken to add to the composite. The analysis was made by a mixed color dithizone method for lead (Procedure III).

### *Fluorides*

Some of the electrostatic precipitator samples were analyzed for both chromium and fluoride. In rinsing such samples from the tubes, slightly alkaline conditions were maintained to avoid possible loss through formation of hydrogen fluoride. Care was exercised that the filter papers used for the filtration were of a type not treated with hydrofluoric acid in their manufacture. After standing for 30 minutes, the rinsings were filtered and aliquots of the filtrate were analyzed for chromium (Procedure I) and fluoride (Procedure IV).

When fluorides were to be determined, the acid treatment of water insoluble material was omitted. The residue and filter paper were placed in a platinum crucible and moistened with sodium hydroxide solution. The paper was burnt off at about 500° C. The residue and ash were mixed with a $MgO$-$Na_2CO_3$ mixture and fused. The water leach of the fusion mass was taken for the determination of chromium (Procedure II) then recombined with the leach residue. The recombined fusion material was then transferred to a fluoride still. Fluorides were separated by distillation from perchloric acid solution and determined colorimetrically with thorium-alizarin reagent (Procedure IV).

When water soluble chromium was not to be determined, the samples were washed from the tubes into a platinum dish with alcohol. The samples were taken to dryness over a steam bath. The dry sample was mixed with

fusion mixture and fused. Chromium and fluoride were determined as previously described for the handling of the water insoluble residue.

### Standard impinger samples

A large number of samples for atmospheric dusts were collected with the standard impinger. Most of these samples were analyzed for hexavalent chromium in the field. Duplicates or aliquots of many of these samples were returned to the laboratory for further analyses. In the field, aliquots were taken volumetrically, whereas in the laboratory the aliquots and total sample were measured gravimetrically.

#### *Chromium*

In the field the samples were analyzed at once by Procedure V for hexavalent chromium. Any compounds of chromium in other valence states were not determined by this procedure.

In the laboratory the sample and bottle were weighed, then shaken thoroughly and about one-half of the entire sample taken for chromium analysis. Then the bottle and remainder of the sample were reweighed. Subsequently, the bottle was weighed empty so that the total sample weight and the exact proportionality of the aliquots could be calculated. The aliquot for chromium was filtered, and the filtrate analyzed by Procedure I for water soluble chromium compounds. The residue was leached with acid as described in the treatment of electrostatic precipitator samples and filtered. The acid filtrate was analyzed for acid soluble chromium by Procedure I. The residue from the acid leach was analyzed by Procedure II. On many of the samples the acid leach was omitted, and this chromium was determined in the fusion of the residue.

#### *Sodium, potassium and calcium*

All impinger samples returned to the laboratory were analyzed for sodium, potassium and calcium. These determinations were made by means of a flame photometer. No treatment of the samples was necessary to prepare them for analysis by Procedure VI.

#### *Sulfate*

Selected impinger samples were analyzed for sulfates by a turbidimetric procedure. An aliquot of about one-tenth of the sample was taken and treated as described in Procedure VII.

### Respirator filter pads

#### *Chromium*

The filter with the sample was macerated in 100 ml of distilled water in a beaker and allowed to steep for 30 minutes at room temperature. The suspension was filtered by suction and thoroughly washed with distilled water until the filtrate totaled almost 500 ml. This solution was made to volume and an aliquot taken for the determination of water soluble chromium compounds by Procedure I.

The paper and sample residue were transferred to a beaker where they were leached several times with dilute nitric acid. The mixture was again filtered under suction and the residue washed with more acid, then with distilled water. Again the washing was continued until the total filtrate was almost 500 ml. This was made to volume and aliquoted. Analysis for acid soluble chromium compounds was made by Procedure I.

The residue from the foregoing acid leaching was transferred to a platinum dish and was ashed in a muffle furnace at 550° C. If the ash were

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rather large it was thoroughly mixed and a small portion weighed into a platinum crucible. This portion was fused with $MgO$-$Na_2CO_3$ mixture and analyzed according to Procedure II to determine the acid insoluble chromium.

The other portion of ash was fused with sodium peroxide, and the chromium determination was made titrimetrically following Procedure VIII. This analysis was used as a check on samples high in acid insoluble chromium.

## Bulk samples

A number of bulk samples of settled dusts, ores, roasts and other dry materials were analyzed for water soluble, acid soluble and insoluble compounds of chromium. The settled dusts were sieved and only that passing a 100 mesh sieve was used for analysis—material coarser than 100 mesh being discarded. Bulk samples other than settled dusts were ground until the entire sample passed through a 100 mesh sieve.

Each sample was thoroughly mixed, and a 100 to 200 mg portion was weighed into a 250 ml Phillips beaker. About 50 ml of water was added and the mixture thoroughly stirred. After standing for 30 minutes it was again stirred, and the suspension was filtered and washed. The filtrate was reserved for the determination of water soluble chromium by Procedure IX.

The filter paper and residue were returned to the original beaker. Ten ml of water and 3 ml of concentrated nitric acid were added. The beaker was placed on a hot plate and the contents brought to a boil for about 10 minutes. The macerated paper tended to cause bumping, so the beaker was removed to a steam bath where it was heated for 1 hour. The suspension was filtered and washed with dilute nitric acid. The filtrate and washings were saved for analysis for acid soluble chromium by Procedure IX.

The filter and residue from the acid leaching were transferred to an iron crucible. The crucible was placed in a cool furnace which was brought slowly to 550° C. to completely burn off the paper. The ash was fused with sodium peroxide and analyzed for insoluble chromium by Procedure VIII.

## Blood and urine samples

Inasmuch as blood and urine specimens were analyzed only for total chromium and there was little or no overlapping of procedures with other samples, the entire treatments and analyses of these samples are given under Procedure X for blood and Procedure XI for urine.

## Spectrographic analyses

Samples of all types taken were analyzed qualitatively and semiquantitatively by emission spectrographic procedures for the metallic constituents. Most samples were dissolved in nitric acid and aliquots of these solutions used for analysis. If a portion of the sample was insoluble in nitric acid, the sample was ashed with nitric acid, evaporated to dryness and a weighed aliquot of the residue used for analysis.

A Bausch and Lomb large Littrow quartz prism spectrograph was used. The samples were excited by a 225 volt, 10 ampere direct-current arc between spectrographically pure graphite electrodes. The elements were identified by their characteristic lines. The semiquantitative results were obtained by comparison of characteristic lines for each metal with the same lines on a series of spectrograms of known amounts of each metal. The comparisons were made visually.

## *Methods of Determination*

In all procedures for chromium, the possible loss due to volatilization as chromyl chloride during the ashing step was prevented by destroying chlorides early in the ashing process with nitric acid, and by avoiding the use of perchloric acid which was found to cause serious losses. Numerous blanks were run for all methods and corrections made. Known samples were run as checks on the procedures.

### Procedure I: Alkaline peroxide method for chromium

A suitable portion of the solution to be analyzed was transferred to a 250 ml Phillips beaker and evaporated to dryness on a hot plate. The residue was moistened with 10 drops of 1:1 sulfuric acid and ashed by the addition of 3 or 4 small portions of nitric acid and heated to fumes of $SO_3$ after each addition. After cooling the ashed sample, the sides of the beaker were washed down with 40 ml of distilled water; then 1 drop of phenol red indicator was added. The solution was neutralized with 4 percent sodium hydroxide and 1 ml added in excess. One ml of 30 percent hydrogen peroxide was added, and the solution was boiled for 15 minutes, during which 1 or 2 more 1 ml portions of peroxide were added to destroy the last traces of indicator and to complete the oxidation of any chromium *(2)*. The solution was then cooled, filtered through a Selas porcelain filter crucible, washed with double-distilled water and made to a volume of 25 ml with double-distilled water. The yellow color of the chromate was read at 373 millimicrons *(3)* in 20 mm cells in a Beckman D. U. spectrophotometer. This reading was compared with a standard curve to determine the chromium concentration.

Knowns prepared from a standard potassium dichromate solution were ashed and carried through the procedure to establish the standard curve. The range was 0 to 150 micrograms of chromium. Larger amounts were determined by dilution of the final solution to bring the color within the range or by starting with a smaller aliquot.

### Procedure II: Fusion method for chromium

Part or all of the ash from the ignition at 550° C. was placed in a platinum crucible with 200 mg of the $MgO\text{-}Na_2CO_3$ fusion mixture (Note 1). The contents of the crucible were thoroughly mixed with a platinum spatula and heated at 900° C. for 30 minutes. After cooling, the crucible was half filled with double-distilled water, and 1 ml of 4 percent sodium hydroxide was added. If a green color resulted from the presence of manganate, a few drops of alcohol were added to reduce the color *(4)*. From time to time a jet of double-distilled water from a wash bottle was directed into the crucible to stir the suspension. After 30 minutes, the contents were filtered through a Selas porcelain filter crucible and thoroughly washed with double-distilled water. The combined filtrate and washings were made up to exactly 25 ml. The transmission was measured in 20 mm cells in a Beckman D. U. spectrophotometer at 373 millimicrons wave length *(3)*. The chromium concentration was then obtained from a standard curve.

NOTE 1.—The fusion mixture *(5)* was prepared by mixing 8 g of magnesium oxide with 2 g of finely divided sodium carbonate and drying at 110° C.

### Procedure III: Lead *(6)*

The composite samples, having already been ashed, were made to a volume of 25 ml and transferred to a 120 ml separatory funnel. To the sample was added 4 ml of 25 percent sodium citrate solution (Note 1) and

1 ml of hydroxylamine hydrochloride solution (Note 2). Six drops of phenol red indicator solution were added and sample was neutralized with lead-free ammonia solution to the first pink coloration. Then 5 ml of potassium cyanide solution (Note 3) was introduced. This was followed by 5 ml of extraction dithizone solution (16 mg/l in chloroform), and the funnel was shaken for 1 minute in a mechanical shaker. The chloroform layer was drained into a clean separatory funnel. The aqueous phase was extracted with successive 5 ml portions of extraction solution until the dithizone color in the last portion remained unchanged. The number of portions used was recorded to estimate the lead content to determine the appropriate aliquot for the final determination. The portions of extraction solution were combined in the funnel. The lead was stripped from the dithizone solution by shaking with 50 ml of 1 percent nitric acid (lead-free). The chloroform layer was separated and discarded. The acid solution was washed with 5 ml of chloroform which was then removed and discarded. Any drops of chloroform on the surface of the aqueous layer were evaporated by blowing across the neck of the funnel. If indicated by the preliminary extraction, an appropriate aliquot of the aqueous layer was transferred to another funnel and made to 50 ml volume with 1 percent nitric acid. To this, 10 ml of ammonia-cyanide mixture (Note 4) and 25 ml of standard dithizone solution (8 mg/l in chloroform) were added. The mixture was shaken for 1 minute, then allowed to separate. A loose plug of clean, dry cotton was inserted in the stem of the funnel, and the chloroform layer was run off into a clean, dry photometer cell. A small amount of the chloroform solution was left in the funnel to make sure that none of the aqueous layer was drawn into the cell. If droplets of water separated on the cell wall, they were removed by carefully pouring the solution into another cell. Photometrically matched test tubes of 20 mm internal diameter were used as cells. The color was read at 510 millimicrons in a Beckman D. U. spectrophotometer, and the lead concentration was calculated from a standard curve.

The standard curve covered the range 0–50 micrograms of lead and was obtained by plotting transmissions against concentrations of a series of known dilutions of a standard lead nitrate solution. The knowns were made to 50 ml and adjusted to be 1 percent nitric acid. They were run through the final extraction as for the samples. Standard dithizone solution was used as the 100 percent transmission standard. A reagent blank was subtracted from all sample values.

NOTE 1.—The sodium citrate solution was rendered lead-free by making it just alkaline to phenol red and extracting with successive portions of dithizone solution until there was no change in the green color of the dithizone. Excess dithizone was removed by repeated washing with chloroform.

NOTE 2.—Twenty grams of hydroxylamine hydrochloride were dissolved in about 65 ml of water. Several drops of m-cresol purple indicator were added followed by concentrated ammonium hydroxide until the solution was yellow. A 4 percent solution of diethyldithiocarbamate in water was added in excess. The reagent was then washed with successive portions of chloroform until no yellow color formed when the chloroform extract was shaken with a dilute solution of a copper salt. Distilled hydrochloric acid was added until the reagent was acid to the indicator (pink). The volume was brought to 100 ml with double-distilled water.

NOTE 3.—Fifty grams of potassium cyanide were dissolved in 100 ml of water and all lead extracted from this solution by shaking with several portions of a strong dithizone solution in chloroform. Any dithizone remaining in the aqueous layer was washed out by shaking with portions of chloroform. The cyanide solution was diluted with double-distilled water to a strength of 10 grams of potassium cyanide per 100 ml.

NOTE 4.—The ammonia-cyanide mixture contained 20 gm of potassium cyanide and 150 ml of distilled ammonium hydroxide (sp. gr. 0.9) dissolved in double-distilled water and made to a volume of 1 liter. This was stored in a cool place.

### Procedure IV: Fluoride *(7)*

The sample was placed in a fluoride still, and 37.5 ml of 70 percent perchloric acid and 0.1 ml of (1 gm/ml) silver perchlorate were added. The mixture was distilled until the temperature reached 137° C.; then steam was introduced and the steam distillation continued, maintaining the temperature of the mixture between 135–140° C. The distillate was caught in a 250 ml volumetric flask to which 1 ml of sodium carbonate solution (11.04 gm/1) had been added. When approximately 225 ml of distillate had been collected the distillation was stopped. The distillate was made to 250 ml with distilled water and mixed. A 100 ml aliquot was taken with a transfer pipet for the colorimetric determination. If this aliquot proved to be too large a suitably sized aliquot was still available for a repeat determination.

The aliquot of the distillate was transferred to an Erlenmeyer flask and 5 ml of alizarin indicator (0.0855 gm of alizarin monosodium sulfonate dissolved in water and made to 1 liter) was added. Sodium carbonate solution was added dropwise until an orange color appeared. The volume of sodium carbonate solution provided a rough measure of the sulfate present; then a correction was made on the basis that 10 mg of sulfate gave the same color change as 0.002 mg of fluoride.

The orange color was discharged by the dropwise addition of 0.3*N* nitric acid. Five ml of thorium nitrate reagent (0.001*M* with respect to thorium nitrate and 1*M* with respect to each of sodium sulfate, formic acid, and sodium formate) was added and the solution mixed by swirling. After standing for a minimum of 30 minutes, the solution was transferred to a Nessler tube and the color compared with a series of standards to determine the fluoride content.

Colorimetric standards were prepared from a standard solution of sodium fluoride (0.01 mg of fluoride/ml). The series of colorimetric standards covered the range from 0.000 to 0.064 mg of fluoride. The appropriate volume of standard solution for each colorimetric standard was made to 100 ml with distilled water and treated in the same manner as the distillate aliquots. Comparisons were made after a minimum of 30 minutes for color development. Standards were usable for several days if protected from air, direct sunlight, and extreme temperature changes.

### Procedure V: Hexavalent chromium

All or an aliquot of the sample solution was placed in a 50 ml, tall-form Nessler tube containing 5 ml of 2*N* sulfuric acid solution. The volume was made to 50 ml with distilled water, and 1 ml of s-diphenylcarbazide reagent *(8)* was added (Note 1). The tube was stoppered and inverted several times to accomplish complete mixing. The color developed was compared visually with a series of standards within 3 to 20 minutes after mixing.

A series of standards of known hexavalent chromium content was prepared with each batch of samples analyzed. A standard solution equivalent to 2.84 micrograms of $CrO_3$ per 5 ml was prepared by diluting 1.7 ml of a 0.01*N* $K_2Cr_2O_7$ standard stock solution to 1 liter. (2.84 mg per cubic foot of air is equal to 0.1 mg $CrO_3$ per cubic meter. The air volumes were measured in cubic feet so this factoral relationship was used.) Appropriate volumes of the dilute standard were placed in 50 ml Nessler tubes and treated in the same manner as the samples. The standard series covered the range of 0 to 7.1 micrograms of $CrO_3$ per sample.

NOTE 1.—Ten grams of phthalic anhydride were completely dissolved in 200 ml of 95 percent ethyl alcohol; then 1.25 gm of s-diphenylcarbazide was added and dissolved.

The solution was made to a volume of 250 ml with 95 percent ethyl alcohol and was stored in a dark bottle, in a cool place if possible. It was found to be usable until an interfering yellow color became apparent in the zero standards.

### Procedure VI: Sodium, potassium and calcium

All impinger samples returned to the laboratory were analyzed for soluble compounds of sodium, potassium and calcium. These determinations were made by means of a flame spectrophotometer. An aliquot of the sample was allowed to settle and the supernatant solution was decanted into a 5 ml sample cup. The cup was positioned in the flame photometer and the solution aspirated into the flame. The intensities of characteristic spectral lines were measured with a photoelectric spectrophotometer. The wave lengths *(9)* used were 590 millimicrons for sodium, 770 millimicrons for potassium and 554 millimicrons for calcium.

A series of standard solutions of each of the three elements was prepared. These solutions were read in the flame spectrophotometer under the same operating conditions as the samples to obtain standard curves for line intensities versus concentrations. The concentrations of the desired elements in the sample were obtained from this curve. Frequent checks of readings with standard solutions were made, and finally each sample was compared individually with the appropriate standard for the final result.

The instrument used was an early model Beckman 10300 flame photometer with a Beckman D. U. spectrophotometer. Natural gas, air and oxygen were used for the flame. Optimum conditions of the combinations of gas pressures were determined for each element by trial with standard solutions. These conditions were then carefully maintained while running the samples. The calcium values were corrected to compensate for interference from sodium. This correction was determined by examining a series of standard solutions of a sodium salt at the wave length for calcium determinations. The correction was small and could be calculated as a percentage of the sodium concentration. Two hundred parts per million of sodium increased the calcium results by one part per million. As a check for interferences, a number of the samples were diluted to one-half and one-fourth their original strengths, and the dilutions were run on the flame spectrophotometer. The results calculated back to the original sample strength checked very well, indicating the absence of significant interferences.

### Procedure VII: Sulfate

The aliquot of the sample was made to a volume of 50 ml with distilled water in a 100 ml beaker, and 2 ml of saturated bromine water was added. The solution was made acid with 1:4 hydrochloric acid as indicated by the appearance of a yellow-brown color and 2 ml of 1*N* hydrochloric acid added in excess. The solution was evaporated to 5 ml and filtered. The residue was washed with distilled water and the combined filtrate and washings evaporated to 5 ml. The sample was transferred with distilled water to a 25 ml glass-stoppered, graduated cylinder; then 1.5 ml of 1*N* hydrochloric acid and 5 ml of 1:2 glycerine-alcohol solution were added. The volume was made to 25 ml and 0.25 gm of solid barium chloride (20–30 mesh crystals) was dropped in. The graduate was shaken until the crystals dissolved. After 40 minutes the sample was mixed again and transferred to a photometer cell. The turbidity was measured by determination of the transmission at 500 millimicrons wave length *(10)*. Matched test tubes with a diameter of 20 mm were used in a Beckman D. U. spectrophotometer.

A standard curve of transmission versus sulfate content was obtained by

running a series of knowns prepared with potassium sulfate through the procedure. The range covered in any aliquot was 0 to 2 mg of sulfate ($SO_4^{-}$).

### Procedure VIII: Titrimetric for chromium *(11)*

The ash was mixed with 2 gm of sodium peroxide in an iron crucible, then covered with an additional 1 gram portion of the peroxide. The mixture was carefully fused for 6 minutes, then was cooled and dissolved in about 200 ml of warm water. The crucible was removed, and a stream of water was used to rinse any adhering material into the solution. After adding 0.5 gm of sodium hydroxide to the solution, it was boiled for 15 minutes, cooled and filtered.

The filtrate was diluted to 250 ml. To this was added 50 ml of 6*N* sulfuric acid and 5 ml of phosphoric acid. The chromate was reduced with 25 ml of standard 0.1*N* ferrous sulfate solution. Three drops of 0.25 percent sodium diphenylamine p-sulfonate solution were added as an indicator. The excess ferrous sulfate was titrated with standard 0.1*N* potassium dichromate solution to a purple end point.

The ferrous sulfate was standardized daily against the dichromate standard.

### Procedure IX: Titrimetric for soluble chromium

The filtrate was ashed with 5 drops of 1:1 sulfuric acid and about four 1 ml portions of nitric acid. To the ashed sample was added 40 ml of distilled water and several drops of phenol red indicator solution. The solution was neutralized to a pink color with 4 percent sodium hydroxide solution; then 1 ml of the caustic was added in excess. The chromium was oxidized with 3 ml of 3 percent hydrogen peroxide, the excess of which was destroyed by boiling the solution for at least 30 minutes.

If the chromate content was low, the solution was filtered and made to an exact volume with 4 percent sodium hydroxide solution. The yellow color was read spectrophotometrically at 373 millimicrons to determine the chromium as in Procedure 1.

If the chromate content was high, the solution was acidified with 50 ml of 6*N* sulfuric acid and 5 ml of phosphoric acid. Excess ferrous sulfate solution was added and back titrated with standard dichromate solution as in Procedure VIII *(11)*.

### Procedure X: Chromium in blood

A specimen of whole blood not larger than 10 grams was weighed into a 125 ml conical beaker, and 1 ml of concentrated, redistilled nitric acid was added. The sample was placed on a hot plate at medium heat and taken to dryness. The addition of nitric acid and evaporation to dryness was repeated with several 1 ml portions of acid and then with successive 0.5 ml portions until ashing was complete. During this procedure the hot plate was gradually raised to full heat, about 400° C. The ash should be a reddish to white color without gray or black coloration.

The ash was dissolved in 0.5 ml of concentrated hydrochloric acid and 1 ml of concentrated nitric acid and evaporated to dryness at medium heat. The sides of the beaker were washed down with 10 ml of distilled water and this evaporated to dryness. Care was necessary that the dry residue was not baked in these steps. The hydrochloric acid was required for solution of the iron oxide in the ash, and tests showed no loss of chromium by volatilization as chromyl chloride when the operation was performed in this manner.

The residue was covered with 25 ml of distilled water, and 2 ml of bromine-sodium hydroxide reagent (6 ml of saturated bromine water per 100 ml of $1N$ sodium hydroxide solution) was added *(12)*. If necessary, $1N$ sodium hydroxide solution was added to make the solution alkaline and precipitate the iron. The solution was gently boiled for 30 minutes and evaporated to a volume of about 4 ml. It was filtered through a Selas crucible and the filtrate caught in a 10 ml volumetric flask, which was also marked at 8.5 ml. The residue was washed with double-distilled water until the volume of the filtrate was just less than 8.5 ml. The filtrate was acidified with 0.5 ml of 25 percent sulfuric acid, and 0.5 ml of a 1.2 percent solution of phenol in double-distilled water was added and the mixture shaken to eliminate excess bromine. Excess bromine must be present as indicated by the color upon the acidification.

After adding 0.5 ml of s-diphenylcarbazide reagent (Note 1) *(8)*, the solution was brought to a volume of 10 ml with double-distilled water and mixed. The color was read immediately at 540 millimicrons wave length. The chromium content was then determined from a standard plot of concentration versus optical density. A Beckman D. U. spectrophotometer was used with a slit width of 0.02 mm. Matched test tubes with a diameter of 20 mm were used as cuvettes.

To prepare the standard curve a series of knowns containing 0–6 micrograms of chromium was prepared from a standard solution of potassium dichromate. These solutions were placed in 125 ml conical beakers, treated with 2 drops of sodium sulfite solution (0.126 gm/100 ml) and evaporated to dryness. The residue was taken up in 0.5 ml of concentrated hydrochloric acid and 1 ml of concentrated nitric acid and continued as with the samples but omitting the filtration.

NOTE 1.—Ten grams of phthalic anhydride were completely dissolved in 175 ml of 95 percent ethyl alcohol and added to a solution of 0.625 gm s-diphenylcarbazide in 50 ml of 95 percent ethyl alcohol. The reagent was diluted to a final volume of 250 ml with 95 percent ethyl alcohol and stored in a brown bottle under refrigeration.

### Procedure XI: Chromium in urine *(13)*

A sample of about 50 ml of urine was measured into a 250 ml borosilicate conical beaker. If the sample contained a residue, this was rinsed out of the bottle with nitric acid and water and added to the urine. A portion corresponding to 50 ml of urine was taken for analysis. The sample was evaporated to dryness on a steam bath. After the addition of 1 ml of redistilled concentrated nitric acid, the sample was placed on a hot plate at moderate heat for a few moments, then swirled to mix and dissolve the residue. Heating was continued until the reaction ended. This procedure was repeated with successive portions of acid decreasing to 0.5 ml meanwhile gradually increasing the heat to about 400° C. Following the formation of a white ash, several additional portions of acid were added to ensure complete ashing. To the ash 2 ml of concentrated nitric acid was added and the acid refluxed on the sides of the beaker for 1 to 2 minutes without boiling off the acid. The sample was evaporated to dryness on a steam bath and was removed promptly to prevent baking of the residue. If the ash did not completely dissolve in the acid, this step was repeated.

The final ash was dissolved in 10 ml of $0.5N$ sulfuric acid, and 0.5 ml of $0.1N$ potassium permanganate was added. The beaker was covered with a watch glass and heated on a steam bath for 30 minutes. If necessary, more permanganate was added to maintain a slight excess as shown by a pink coloration of the solution. A 5 percent solution of sodium azide in double-

distilled water was added dropwise at the rate of one drop every 10 seconds, with swirling after each drop, until the permanganate color was discharged. Sufficient sodium azide solution was used to destroy any brownish tint, but an excess was avoided. After destruction of the permanganate, the sample was removed immediately from the steam bath and placed in a tray of cold water. Under these conditions, no chromate was reduced by azide. If necessary, the solution was filtered through a Selas crucible to remove insoluble materials. It was then transferred to a 25 ml volumetric flask with double-distilled water. One ml of s-diphenylcarbazide reagent *(8)* (this is the same reagent as used in Procedure X) was added. The solution was thoroughly mixed by shaking and allowed to stand one minute for development of the color. Then 2.5 ml of 4*M* sodium dihydrogen phosphate solution was added, the volume was made to 25 ml with double-distilled water, and the solution was thoroughly mixed.

The color was read at 540 millimicrons at a slit width of 0.02 mm in a Beckman D. U. spectrophotometer. Matched test tubes of 20 mm diameter were used as cuvettes, a special holder having replaced the usual cuvette holder of the instrument. The chromium concentration in the sample was calculated from a prepared standard curve of optical density versus concentration. Distilled water was used as a reference blank.

Standard potassium dichromate solution was reduced to trivalent chromium with sodium sulfite and dilute nitric acid. Excess sulfite was destroyed by evaporating almost to dryness and adding 0.5 ml of nitric acid and evaporating gently to dryness. The residue was dissolved in 1 ml of nitric acid and made to desired volume with distilled water. Suitable volumes of this trivalent chromium standard were measured into conical beakers and carried through the procedure. The optical densities of these knowns were plotted against chromium content covering the range of 0–16 micrograms of chromium. A trivalent chromium standard was used as a check on the oxidation reaction although standard dichromate was also used with good agreement.

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